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RESEARCH REPORT

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16. Abstracts: DDT was selected as the compound for this study due to paucity of data regarding environmental effects of other classes of pesticides. Human exposure to pesticides was examined from two major pathways; the direct pathway which deals with direct uptake from primary sources of pesticide release, and the indirect pathway which involves human exposur by translocation through air, water, or food. Reductions in accidential poisonings could be made by limiting the total toxicant contents of home packages of pesticides, and means to reduce deaths from aerial applications warrant further study of this method of application. The control of human burden of pesticides by control of food residues is only partially effective since evidence on pesticide distribution in the environment suggests that about half of the intake is from inhalation of insecticide aerosols or dust laden with insecticides. There is presently no evidence that pesticide exposure directly shortens human life. Fish and birds are more sensitive to pesticides in the environment. Fish lack the basic microsomal oxidase enzyme systems which detoxify pesticides and birds appear to be very sensitive to the sterodal hormone mimicking effects of the chlorinated hydrocarbons.

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FINAL REPORT

on

TECHNICAL, INTELLIGENCE, AND PROJECT INFORMATION SYSTEM FOR THE ENVIRONMENTAL HEALTH SERVICE VOLUME IV. PESTICIDES MODEL CASE STUDY

to

U. S. DEPARTMENT OF HEALTH,
EDUCATION, AND WELFARE
ENVIRONMENTAL HEALTH SERVICE

June 29, 1970

Contract No. CPS-69-005

by

J. K. Yoss, J. W. Blaylock, M. J. Schneider, L. C. Schwendiman,
C. J. Touhill, Jr., W. L. Templeton, R. E. Wildung, and D. B. Menzel

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FOREWORD

This report is one of five volumes that were prepared for the Environmental Health Service under Contract Number CPS-69-005. The objectives of this report were to examine research and development program planning needs within the EHS and to develop a management assistance system for the EHS. Since a well-organized and readily accessible information base is essential to planning, existing information capabilities and facilities within EHS were surveyed and an environmental health information network was devised. Two model case studies for the environmental stressors, lead and persistent pesticides, were performed to serve as examples during the project and to exercise the elements of the management assistance system.

The reports in this series are:

Volume I. EHS Management Assistance and Planning

Volume II. EHS Information Network Analysis

Volume III. Lead Model Case Study

Volume IV. Pesticides Model Case Study

Volume V. Directory of EHS Information Facilities With Selected Supplementary Resources

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MANAGEMENT SUMMARY

In order to demonstrate the utility of the methodology described in the preceding portions of this report, two model cases were examined. This volume deals with a case study of pesticides. Because of numerous technical difficulties, we found that DDT was the most reasonable model compound for study. This decision was dictated primarily because of paucity of data regarding environmental effects of other classes of pesticides. DDT is useful because it has been in widespread use longer than any other material and has demonstrated a wide interaction with all of the potential transport pathways for man-made chemicals in the environment. Because we have found it necessary to restrict ourselves to DDT, one major conclusion is obvious: sufficient information is not now available about the quantitative mechanisms of redistribution of pesticides in the environment. Specifically, major deficiencies exist in the aerial transport and uptake of pesticides by man, the environmental reservoirs of pesticide storage, the chronic effects of pesticides upon man and wildlife (from the molecular view rather than simply mortality estimates) and the ultimate fate of pesticides.

In our study we found it useful to divide human exposures into two major pathways. We have chosen to call the first the direct pathway, which deals with the direct uptake from primary sources of pesticide release. Primary sources were manufacture and application. Human exposure was by inhalation. Dermal uptake and ingestion are secondary features. Accidental poisonings remained about the same, even though the frequency of application and the acute toxicity of the new pesticides has increased. These trends suggest that a very credible job of education is being done at the industrial level. Since most of the deaths from accidental poisoning were children, further reductions could be made by reduction of the total toxicant contents of the home package to below the child-lethal dose, where possible, and elimination of those chemicals which can not be used within the safety margin (e.g., substitution of carbamates and pyrethrians for organophosphate insecticides). Prescription and dispensing pesticides by trained professionals would also reduce accidental deaths. Aerial application accounts for the largest number of deaths and either elimination or control of this method appears worth study.

The second pathway, the indirect pathway, involves human exposure by translocation through the air, water, or food. While more complicated to consider and model, the indirect pathway involves the total biosphere. The general population of the U. S. is exposed by this means. Serious conflicts exist in the data as to which indirect pathway leads to the major store of pesticides in man. On a worldwide basis, the persistent pesticide content of man is remarkably constant. Within the U. S., there are, however, significant racial and geographic differences. Such differences are difficult to explain if food is the major transport pathway to man. The southern population and the southern Negro have greater levels of pesticides than the northern equivalent populations. Together with some indirect evidence from residues in animals, it is likely that only 50 percent of the body burden is from food, and the remainder may come from inhalation of insecticide aerosols or dust laden with insecticides. If these observations are correct, then control of the human burden of pesticides by control of food residues, as is now practiced, is at best only partially effective.

There is, at present, no evidence to attribute a direct shortening of human life to pesticide exposure. There are suggestions that large amounts of pesticides will increase the frequency of certain tumors in animals. Whether or not pesticides induce cancers in man cannot be settled at present, because there appears to be a dose dependence in cancer induction. Whether tumors or cancers in man can be induced at the low levels at present in foods is not known. These studies suffered from the relatively small numbers of all toxicological experiments and the short term of exposure.

In any event, man is not the animal in the environment most sensitive to pesticides. Animals of the aquatic environment are particularly sensitive because the translocation pathways are highly conservatory of persistent compounds. Persistent compounds become concentrated rather than diluted as residues pass up the food chain. Fish and birds are inherently more sensitive to pesticides than mammals. Fish appear to lack the basic microsomal oxidase enzyme systems found in all mammals which detoxify many pesticides. Birds appear to be very sensitive to the steroidal hormone mimicking effects of the chlorinated hydrocarbons. Hormonal effects in man are not known.

Only the soil system appears to be a "safe" repository for pesticides. Conversion to non-toxic metabolites can also be promoted in the soil by anaerobic conditions. The aquatic ecosystem can be protected from pesticides by modifications of agriculture practice to ensure that pesticides are retained and degraded within the soil system. Little hazard to man through the groundwater exists.

Finally, while it is possible to model crudely the distribution of DDT in the environment and to suggest ways to reduce the environmental burden of DDT, these efforts will have little direct effect on human health. Advances in protection for aquatic birds and fish can be accomplished. This study suggests that regulation of pesticides on the basis of their hazard to human health is less important than regulation of the basis of their hazard to wildlife. Human health will most likely be improved rather than degraded by emphasis on wildlife since regulations based upon harmful effects to wildlife will inevitably reduce human exposure. This does not mean that careful evaluation of pesticides for human health hazards is unnecessary. On the contrary, more information is needed. Rather, this study suggests that the rate of application and hence long-term residual levels in the environment are best regulated with more concern to wildlife survival. The margin of safety between levels evoking chronic effects in man and environmental levels will increase by this procedure. Man will hence be "safer".

TECHNICAL, INTELLIGENCE, AND PROJECT INFORMATION SYSTEM FOR THE ENVIRONMENTAL HEALTH SERVICE VOLUME IV. PESTICIDES MODEL CASE STUDY

by

J. K. Yoss, J. W. Blaylock, M. J. Schneider, L. C. Schwendiman, C. J. Touhill, Jr., W. L. Templeton, R. E. Wildung, and D. B. Menzel

OBJECTIVE

The pesticide model case study was undertaken to determine how effectively the management assistance system can function as structured during the project, pinpoint areas where improvement of the system is needed, provide examples of the types of information on the environmental stressors that are currently available from information sources, and to indicate in a preliminary manner the additional research and development and other projects that should be considered by EHS in light of the present situation. All the elements of the management assistance system were to be exercised, which includes the stressor matrix, the rating system, the mathematical model, and the information network, through the pesticide model case study.

INTRODUCTION

Pesticides have become widely distributed in the environment. Great concern has arisen recently over the effects of pesticides in the environment. Much of this concern has been directed at the widespread distribution of persistent pesticides, such as DDT, in the environment. The chlorinated hydrocarbons (the most persistent pesticides) are implicated in the reproduction and survival of nontarget species. Extensive field data and controlled-experiment results indicate that certain birds, fishes, and insects are especially vulnerable to these pesticides.[1-4] It has recently been suggested that DDT even affects photosynthesis in the oceans.[5] DDT and other persistent pesticides are also found in man.[6]

The general case of pesticidal impact on man and the environment was examined and this led to a detailed discussion of two aspects of pesticide usage. Direct exposures to a limited group of individuals are evident through a pathway referred to in this study as direct interactions. A second pathway, indirect interactions, exposes the whole population and the environment. Because of the scarcity of data in many instances, DDT has become the specific model compound for this study. Some considerations of the divergence to be expected from other compounds in agricultural usage will be made at the conclusion.

TOXICITY OF PESTICIDES TO MAN

Pesticides are developed to be toxic to certain forms of life. For example, fungicides are developed to be toxic to fungi, insecticides to insects, herbicides to plants, etc. Unfortunately, at sufficiently high doses, pesticides can cause adverse effects on nontarget forms of life, even man. Man has been fatally poisoned by almost all of the major pesticides. There is little doubt that almost all the pesticides can cause illness in man when exposed under certain conditions.

The chlorinated hydrocarbons are one of the most important groups of synthetic organic pesticides. They are widely used and are toxic to man. They are not easily biodegradable and persist in the environment for relatively long periods of time. When chlorinated pesticides enter man, they are not readily metabolized but are stored in the body fat and other organs of the body.

The chlorinated hydrocarbons are absorbed into the body through the respiratory tract, digestive tract, the skin, or mucous membranes.

Acute Exposure

Chlorinated hydrocarbons affect the central nervous system. The mechanism of this action has not been determined. The symptoms of exposure in mild cases are characterized by headaches, dizziness, gastro-intestinal disturbances, numbness, and weakness of extremities, apprehension, and hyperirritability. Larger doses induce muscular tremors finally leading to convulsions, cardiac or respiratory failure, and death. Pesticides responsible for accidental deaths in the United States and the corresponding number of deaths for 1956 and 1961 is shown in Table 1. Table 2[8] shows the distribution of deaths according to age and route of exposure. Of the 119 deaths in 1961 related to pesticides, 111 were identified as coming from known materials. Fifty-seven percent of the cases were in children under 10 years of age.

It is interesting to note that control of the acute intoxication has been attempted by many means. The success of the current control efforts can be evaluated by the fact that the incidence of fatal poisoning in the U. S. has held constant, one person per million, despite the increase in availability and use of pesticides in the last 25 years.

Much data is available on the dosages of DDT able to produce illness in man. A single ingestion of 10 mg/kg produces illness in some but not all subjects even though no vomiting occurs. Smaller dosages generally produce no illness.[10] The estimated oral fatal dose to man is 500 mg/kg, with death occurring within 2 to 24 hours.[11]

Volunteers have ingested about 35 mg/man/day DDT for as long as 21 months without producing any clinically defined effects. This dosage of DDT is about 1000 times the dosage the average person receives in the diet.[12] The minimum daily DDT dosage producing acute illness is not known. However, from animal experiments, it has been suggested that some individuals will become mildly ill at a dose ranging from 2.5 to 5 mg/kg/day.[10]

TABLE 1. IDENTITY OF PESTICIDES RESPONSIBLE FOR ACCIDENTAL DEATHS IN THE UNITED STATES IN 1956 AND 1961[8]

Pesticide	1956	1961
Inorganic and Botanical Solid and Liquid Pesticides		
Arsenic	54	29
Thallium	8	2
Mercury		1[a]
Phosphorous	21	12
Fluorides		6
Sodium chlorate	1	
Calcium polysulfide		1
Boric acid	1	
Cyanide (solid)		3
Copper oleate mixed with tetrahydronaphthalene	1	
Strychnine	3	1
Nicotine	4	3
Pyrethrum	1	
Camphor	1	
Rotenone, copper, and sulfur	1	
Subtotal	96	58
Organic Phosphorus Insecticides		
Diazinon	2	1
Demeton	1	
Malathion	3	3
Methyl parathion		3
Parathion	11	15
Mevinphos		1
TEPP	2	
Unspecified organic phosphorus insecticides	1	1
Subtotal	20	24
Gases and Vapors (E890-E895) (Pesticides Only)		
Carboxide gas[b]		1
Cyanide gas	11	3[a]
Ethylene dibromide		1
Mercury	2	
Methyl bromide	3	
Sulfur dioxide	1	1
Subtotal	7	6
Grand Total	152	111

(a) Diagnosis of pesticide poisoning open to serious question.
(b) Mixture of ethylene oxide and carbon dioxide.

TABLE 2. DISTRIBUTION OF ACCIDENTAL PESTICIDE DEATHS IN THE UNITED STATES IN 1961

Parameter	Age Group <10	10-19	20-69	≥70	Total
Occupational					
Yes	0	4	12	1	17
No	57	4	24	2	87
Unknown	0	0	7	0	7
Total	57	8	43	3	111
Route of Exposure					
Oral	47	3	27	3	80
Respiratory	1	1	3	0	5
Dermal	1	0	0	0	1
Combined	5	3	9	0	17
Unknown	3	1	4	0	8
Total	57	8	43	3	111

There is no well-described case of uncomplicated DDT poisoning. DDT relative to other pesticides must be regarded as only nominally acutely toxic to man. Other pesticides, such as parathion, are regarded as extremely toxic to man. If a child eats as little as 2 mg of parathion, the result is death. There were no known cases of deaths caused by acute intake of DDT through 1963.[10]

Chronic Exposure

While the cases of acute exposure to man are relatively clear cut and adequate data exists, little is known about the effects of long-term exposure of humans to comparatively low levels of many pesticides in the environment and in the body itself. Tables 3 and 4 show that humans now carry in their body fat from 10 to 20 ppm chlorinated hydrocarbon insecticides and their metabolites.[13]

DDT is the oldest chlorinated hydrocarbon. It has been in the environment for over 20 years. As far as can be determined, DDT has no detectable chronic effect on man. Man has ingested DDT 1000 times the average dosage with no detectable effects except that more DDT was stored, metabolized, and eliminated at a higher rate.[14] Figure 1 shows the lack of effect of the advent of DDT on death rate trends for five diseases.[15] There is no sharp deviation from the trend from 1945-1965, during the period when DDT came into wide use.

The inability to detect any adverse effect of DDT in man does not mean that there is none. Because of the ubiquitous nature of DDT, it is now impossible to find a group of DDT-free individuals. Since there are no DDT-free humans, there is no way of observing any slight change in man caused by DDT. Recent research, however, has shown that DDT at low-level doses could be degrading to man's health.

TABLE 3. CONCENTRATION OF DDT-DERIVED MATERIAL IN BODY FAT

Country	Year	No. of Samples	Analysis Method	DDT[a], ppm	DDE as DDT, ppm	Total[b] as DDT, ppm	DDE as DDT, % of total
United States	1961-62	28	Colorimetric	3.7	6.9	10.6	65
			GLC[c]	2.4	4.3	6.7	64
France	1962-63	5	Colorimetric	3.1	6.5	9.6	68
			GLC[c]	3.5	5.3	8.8	60
India (Delhi area, civilian)	1964	24	Colorimetric	18	12	30	40
			GLC[c]	14.3	12.9	27.2	47
India (other cities, military)	1964	11	Colorimetric	7	4	11	36
			GLC[c]	4.7	7.1	11.8	60

(a) Includes p, p'- and o, p'-DDT.
(b) Includes all detected isomers of DDT and DDE, but not DDD.
(c) Gas-liquid chromatography.
Source: Hayes, W. J., "Monitoring Food and People for Pesticide Content", in "Scientific Aspects of Pest Control", Publication 1402, National Academy of Sciences - National Research Council, Washington, D. C., 1966.

TABLE 4. AVERAGE CONCENTRATION OF CHLORINATED HYDROCARBON PESTICIDES (OTHER THAN THOSE DERIVED FROM DDT) IN BODY FAT OF THE GENERAL POPULATION OF VARIOUS COUNTRIES[a]

Country	Year	No. of Samples	Storage Level in Body Fat, ppm			
			BHC Isomers	Dieldrin	Endrin	Heptachlor Epoxide
United States	1961-62	28	0.20	0.15	(b)	(b)
United States	1962-63	282	0.57	0.11[c]	nd	(b)
United States	1964	64	(b)	0.31	<0.02	0.10
United States	1964	25	0.60	0.29	<0.03	0.24
England	1961-62	131	(b)	0.21	(b)	(b)
England	1963-64	65	0.42	0.26	(b)	<0.1
England	1964	100	0.02[d]	0.21	<0.02	<0.01
France	1961	10	1.19	(b)	(b)	(b)
India	1964	35	1.43	0.04	nd	nd

(a) All analyses were carried out by gas-liquid chromatography.
(b) The specific pesticide was not tested for, or at least such testing was not mentioned in the published paper. If the pesticide was looked for but not detected, the result is shown as less than the sensitivity of the method used when this value is known; or, if the limit of sensitivity is not known, the result is simply shown as not detectable (nd).
(c) Only 64 samples were tested for dieldrin content.
(d) Only 20 samples were tested for BHC content.
Source: Hayes, W. J., "Monitoring Food and People for Pesticide Content", in "Scientific Aspects of Pest Control", Publication 1402, National Academy of Sciences - National Research Council, Washington, D. C., 1966.

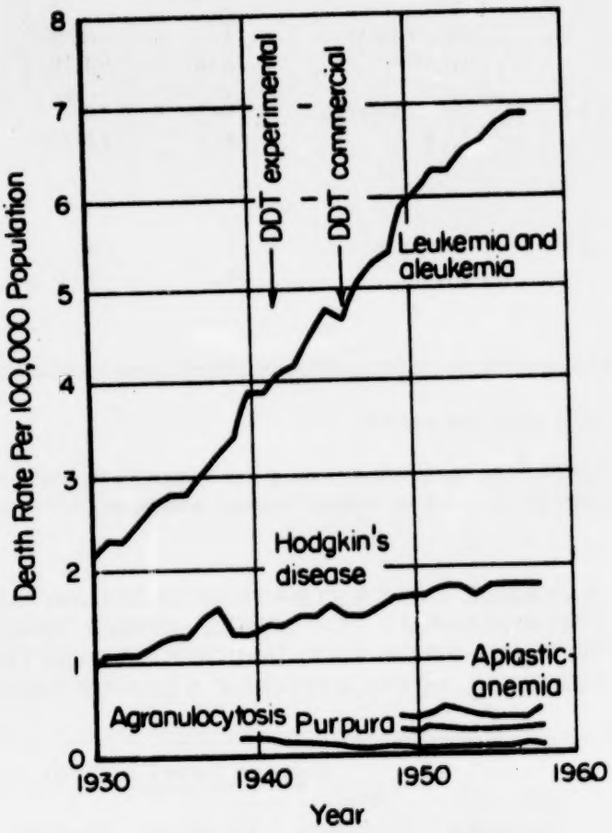


FIGURE 1. LACK OF EFFECT OF ADVENT OF DDT ON DEATH-RATE TRENDS FOR FIVE DISEASES

Curves show no marked changes in trends in death rates from leukemia and certain diseases of the blood following the introduction of DDT, which has sometimes been suggested as a cause of all of the diseases shown.

DDT and some other pesticides accelerate their own metabolism in the body. DDT increases the activity of enzymes in the liver, and these enzymes in turn metabolize DDT faster. It has recently been demonstrated that this enzyme activation occurs in some birds. Eventually, the birds reproductive processes are disrupted and the species declines. Whether a similar effect will be seen in man is not clear.

Tumors have been induced in animals when exposed to DDT.[17, 18] The possibility exists that DDT can cause cancer in humans on long exposure in the environment. Before conclusions can be drawn, more knowledge is needed on chemical carcinogens and how they cause cancer.

Some physicians have expressed concern over the significance of DDT in subacute levels from two standpoints. One is the individual susceptibility of DDT and other poisons. The other is the carcinogenic properties of halogenated hydrocarbons.[19] Doctor Malcolm M. Hargraves[19] contends that in his clinical practice he has seen many cases which are convincing to him that airborne organic compounds in the environment precipitate serious illnesses and eventual death in people who are particularly sensitive to these agents. Other witnesses[19] present case histories to bear out this contention. DDT is not singled out. Because of individual susceptibility, it becomes difficult, if not impossible, to assess the impact of very small quantities in the air breathed by a large population. Much needs to be learned concerning this very important subject of individual susceptibility. There is very little or no statistical data to prove that very low levels of DDT may precipitate a serious illness.

There is no conclusive evidence showing that long-term exposure to pesticidies at current concentration in the U. S. is causing any chronic effects on man; however, the possibility exists.

THE DIRECT PATHWAY

In the manufacturing, transportation, formulation and use of all pesticides, a potential exists for direct accidental or inadvertent exposure of people to the toxicants. The only reported physiological effect of DDT on man has been through direct contact with the material. Air is the main vehicle through which the pesticide reaches man directly, although deaths have occurred from ingestion of pesticides and their diluents. All potential direct routes are examined, even though little information was found about the significance of some particular routes or sources.

Distribution From Primary Sources

The first interaction between man and a pesticide arises in the manufacturing process. The complex processing steps eventually lead to the technical product. Packaging is the end step in this process. Further operations bringing man in contact with these materials are formulation and transportation. The direct pathway will be created during all working hours for occupational workers and the consequences will hinge on the protective measures instituted and followed. In addition to the nominal day-in/day-out contact with these chemicals, there exists the potential for serious accidents giving

rise to extensive clouds of fine particulate material or, when fires are involved, vapors of the parent compound and toxic pyrolysis compounds.

Following formulation and transport, the pesticide may be stored for long periods. Though not a cause of great concern, serious contamination of foodstuffs by insecticides nevertheless has occurred due to cross contamination during storing or mislabeling at some time during storage.

The end use of the pesticide creates the most important and critical source for the major direct pathway. Not only the applicators and others whose work brings them into frequent contact with these poisonous materials but members of families, who from poor control or accident, are poisoned through direct contact.

Figure 2 is a diagram which calls attention to the sources of DDT giving rise to the direct contact with man. Also shown on the diagram are the routes to man and the ways by which the pesticide is introduced into the body.

The arrows upward from the source blocks call attention to the acute or chronic releases to the atmosphere in a manner such that great dilution occurs due to atmospheric processes. This airborne atmospheric pool of DDT provides a direct interface when people in a region breathe this air, but, as will be shown in the discussion to follow, this source and pathway is of much lesser importance than other routes to man.

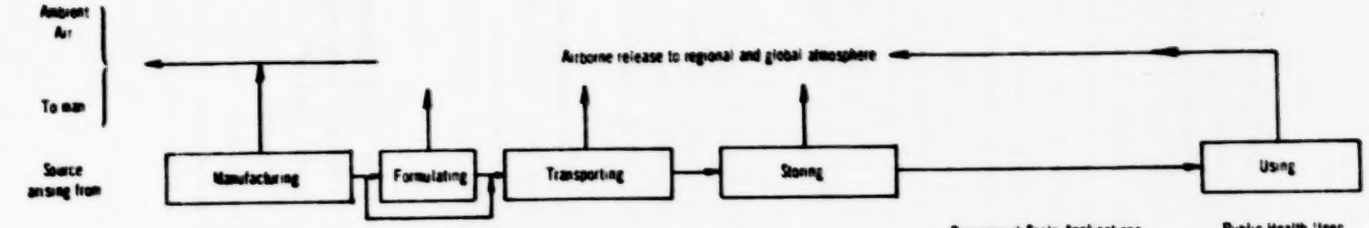
Significance of the Direct Pathways

The Ubiquitous Airborne DDT "Background"

The widespread and prolonged use of DDT coupled with its persistence gives rise to concentrations of the compound in virtually every part of the world. Sampling and analysis at the very low concentrations are difficult and only limited data are available. Some observations can be made regarding the physicochemical behavior of DDT to help determine the significance of this source and interface with man.

All of the sources shown in Figure 2 can, and likely do, contribute to the atmospheric reservoir of DDT. The compound is used as very fine powder and in various emulsions and solutions. Particles and droplets larger than a few microns will deposit on the ground and on water surfaces rather quickly. These secondary sources can again introduce the compound to the air. DDT co-distills with water at 25 to 35 C[20], and contaminated water could be a steady source of airborne DDT. Fires involving public carriers or storage warehouses and disposal of containers introduce fine aerosols containing DDT into the atmosphere.

The distribution of airborne materials depends dominantly on the local meteorological conditions, including rainfall and the particle size. Other factors will include the stability in light, oxygen, and heat in the atmosphere. Even though these effects on persistence are not individually known with much precision, measurements of airborne DDT in urban communities support the conclusion that the "background" airborne concentration is of minor concern compared with other sources and pathways to the public at large. A study was made of DDT air concentrations in six urban communities, each of which was in the center of an agriculture or fruit-growing area.[21] Crops were sprayed



Routes to and "into" man		Manufacturing	Formulating	Transporting	Storing	Using: Commercial Scale Applications	Using: Public Health Uses	Using: Personal and Home Uses
				(Accidents involving spills, fires, cross contamination.)	(Accidents involving spills, fires, cross contamination.)	a. Field crops, orchards b. Forestry c. Greenhouse operations d. Home pest control businesses e. Dry cleaning operations f. Grain sterilization g. Animal and poultry insect control h. Control in food processing plants i. Food preparation and serving industries (bakeries, restaurants)	a. Mosquito control	a. Insect control, gardens, lawns b. "Moth proofing" clothing c. Body parasites d. Paints
	Via air	1. Inhalation by workers of airborne particles, vapors. 2. Dermal contact, sprays, touching gloves to face, etc.	(Same)	1. Inhalation by people in vicinity. Powders, vapors of pesticide or decomposition products. 2. Dermal contact. Vehicle driver, people in vicinity. Sprays, splashes.	1. Inhalation by people in vicinity. Powders, vapors of pesticide or decomposition products. 2. Dermal contact.	1. Inhalation a. By occupational worker b. By others in vicinity (empty containers, accidental release) 2. Skin Contact a. By worker – sprays, dusting, cigarette smoking b. By user of product treated (clothing treated, for example) c. Worker's family – babies touching clothes, cars, etc.	1. Inhalation a. Occupational worker b. Home occupant c. Bystander 2. Skin Contact a. By worker	1. Inhalation a. Occupant b. Bystander, neighbors 2. Skin Contact a. Direct spray b. Clothing
	Via water	1. Drinking contaminated water on premises.	(Same)	1. Possible accidents near river supplying drinking water. Accidents on bridges. Seriously contaminated water supplies.		1. Accidental use of used containers for water supply		1. Accidental drinking by children. 2. Reuse of containers.
	Via food	1. Possible contamination of food eaten on premises. 2. Contamination of cigarettes, confections.	(Same)	1. Gross contamination of food products transported in the same carrier.	1. Gross contamination of food products. Reuse of containers, storage facilities, etc.	1. Contamination of treated food products. 2. Contamination of lunches on job. 3. Contamination transfer from hands to cigarettes.		1. Contamination of cooking utensils. 2. Use of improper containers with no labeling or poor labeling.

FIGURE 2. THE DIRECT MAN-PESTICIDE INTERFACE

heavily during the growing season and samples were collected during the same period. Concentrations found ranged from less than detectable to 20 x 10^{-9} g/m^3. The Threshold Limit Value for DDT is 1.0 mg (1 x 10^{-3} g)/m^3, although the TLV appears to be of questionable value in chronic exposure. The author leaves some doubt as to the validity of sampling and analytical measurement; it nevertheless is apparent that the levels found do not contribute significantly to the burden of the population at large by direct contact. In a second study, the DDT associated with airborne particles in Pittsburgh was measured. Detectable but low (up to 1 x 10^{-9} g/m^3) concentrations of DDT were found associated with particles.[22]

Manufacturing Processes

The manufacturing processes for DDT and other pesticides are complex chemical engineering operations requiring close contact with the product, particularly near the end of the process where grinding and bagging operations occur.[23] Manufacture of DDT on a commercial scale in the United States began about 1945-1946; thus, over 20 years of exposure has occurred to some workers. The literature on exposures and lost-time incidence reveals no particular significance of DDT processing as a hazardous occupation. Workers in a large DDT plant producing some 6 million pounds of technical product a month were examined intensively by a physician.[24] These workers had a minimum of 5 years at their jobs and some had 9 to 19 years. The physician reported that the clinical findings of his examination did not differ significantly from those expected in a group of similar age and socioeconomic status but with no exposure to DDT. The fat samples taken from these workers showed up to several hundred ppm of DDT. It was estimated that the occupational intake was of the order of 17 to 18 mg per day. (This is compared with the estimated average general population intake of 0.04 mg per day.)

Similar conclusions were reached regarding occupational disease in a DDT factory (700 tons/year) in England.[25] Although DDT was found in "toxic concentrations" in the grinding section, there was no indication of occupational disease. Workers were subsequently provided with respirators and underwent periodical medical examinations.

A study was undertaken to determine whether spraying Dutch Elm trees posed any threat to the workers.[26] Volunteers ingested 35 mg of DDT each day for 18 months with no apparent deleterious effects and the author concluded that the program for controlling Dutch Elm disease could be safely undertaken. Again, these data refer only to acute intoxication.

A comprehensive, consolidated review of occupational exposure in DDT-formulating plants apparently has not been prepared, or at least the literature reviewed revealed no such study. The information available supports the observation that good industrial-hygiene practices could prevent virtually any manifest occupational disease. Chlorinated hydrocarbons affect the central nervous system. Whether or not the rather heavy long-term exposures to airborne DDT particles generated in chemical processing and formulating have affected in some subtle way the mental acuity or other mental response or could cause life shortening was not determined.

DDT is one of the least toxic pesticides, and observations therefore must not be applied to other pesticides, many of which are much more toxic than DDT. In Figure 2, modes of exposure other than inhalation are noted for those working in the manufacture of a pesticide. While of even less consequence to workers in a DDT plant, the routes of skin contact and ingestion will become important for the more toxic compounds such as organophosphates and carbamates. Much more critical industrial hygiene and medical protection would be demanded for these more toxic materials.

Formulating

DDT may be blended with inert powder and other insecticides prior to use, or it may be suspended in oil and water as an emulsion of widely varying concentrations. Formulating operations pose no more of a problem than the manufacturing processes. When DDT is combined with more toxic materials, care must be exercised. The literature review revealed no studies specifically concerned with formulating.

Transporting

Delivering the product to the point of use presents the potential of collisions, fires, derailments, and other energetic stresses which may release large quantities of the poison carried to the vicinity of the accident. It was estimated that 80 percent of the tonnage is carried by trucks and motor carriers, 17 percent by rail and 3 percent by water.(27) Transportation accidents, as judged by the reports located, have been of virtually no consequence with respect to DDT. A potentially serious spill of parathion did occur near Blythe, California, which received considerable publicity, but even with this much more toxic chemical there were no fatalities.(27) Such an accident happening in a large metropolitan area may have much more serious consequences.

One aspect of concern in transporting pesticides and other poisons is that of cross contamination of foodstuffs with these poisons. A truck used for pesticide may subsequently be used for hauling grain or food products. No such incidences were disclosed insofar as DDT was concerned; however, in seven incidents a total of 100 persons, mostly children, died of poisoning and more than 1000 were poisoned but survived.(27) In most incidents the commodity contaminated was food, but in two cases the commodity was clothing or bedding.

Storage

Storage of DDT and other poisons provides an opportunity for fires and cross contamination of other materials. Twelve major fires occurred in 1954-1967 in buildings in which pesticides were stored.(28) Several of the fires involved mixed pesticides including chlorinated hydrocarbons. Property loss ranged from $50,000 to over $400,000 per fire, and at least 127 firemen, volunteers, and bystanders were poisoned in these fires. Special problems were presented in the safe disposal of the remaining pesticides.

Many instances have been documented in which poisoning has occurred because of unmarked or mislabeled pesticide containers. In a 15-month period six California workers died because they drank pesticides, mistaking them for water or other beverages.(29)

A study of 62 pesticide accidents in various parts of the United States between 1963 and 1965 showed them to arise from the following causes:

Improper storage or security of pesticides	16
Pesticide placed in different containers	13
Pesticide spilled on clothing	8
Improper disposal of "empty" containers	7
Rain runoff from treated field or storage area	2
	46

Thus, 46 of the 62 accidents involved improper storage.

The significance of improper storage as a source for direct interface with man cannot be overemphasized. Although DDT has not been the chemical of greatest concern because of its lesser toxicity than other pesticides, it has been involved in several incidents involving storage.

Proper storage and fire-control systems can be instituted that greatly reduce the potential for ingestion and inhalation. Like other industrial hazards, the ultimate reduction will come through good training and insistence on following prescribed practices.

Use of Pesticides

Use of pesticides has become so widespread in the United States that nearly every person has access to one or more formulations. While a large fraction of pesticides is used in agriculture, forestry, and other large-scale applications, over $110,000,000 worth of pesticides are used each year around homes, representing about 5 percent of total U. S. production.[30] Thus, widespread opportunity exists for accidents involving these poisonous materials. The interface with man is achieved in a variety of ways, as pointed out in Figure 2.

Statistics available on deaths and sickness due to pesticides seldom permit a direct determination of the discrete chemical involved, and the significance of DDT as a agent must usually be inferred, or, in the extreme pessimistic point of view, all pesticides problems could be ascribed to DDT. The data coming to attention were usually presented with qualifications as to completeness and accuracy.

The State of California appears to have maintained the best records on occupational deaths and disease from pesticides, and from these data one can draw some conclusions regarding the occupational and other accidental cases of pesticide and other agricultural-chemical poisoning (Tables 5 & 6)[31, 32] From 1000 to 1300 occupational diseases due to pesticides are recorded each year. Reports of occupational disease showed an increase from 377 in 1953 to 1,013 in 1963. About two-thirds of the incidents were in agriculture. About one-half of the reports involved skin disease. During a 10-year period, 1953-1962, DDT and other chlorinated hydrocarbons accounted for about 4 percent of the occupational poisoning cases. Over 70 percent of systemic poisoning cases and 28 percent of all occupational diseases reported were attributed to organophosphates. In 1961, 14 nonfatal poisoning cases were attributed to DDT and related compounds. In 1961, 15 lost-time days were reported for occupational disease caused by use of DDT, chlordane, lindane, and kelthane.

TABLE 5. OCCUPATIONAL DISEASES BY INDUSTRY ATTRIBUTED TO PESTICIDES AND OTHER AGRICULTURAL CHEMICALS CHEMICALS IN CALIFORNIA, 1953-1963[31]

Year	All Industries	Agriculture	Other Industries[a]
1953	377	277	100
1954	391	248	143
1955	531	326	205
1956	789	464	325
1957	749	434	315
1958	910	599	311
1959	1,093	782	311
1960	975	668	307
1961	911	578	333
1962	827	545	282
1963	1,013	746	267

(a) Includes service, construction, manufacture, government, etc.

TABLE 6. OCCUPATIONAL DISEASES BY CHEMICAL COMPOUND ATTRIBUTED TO PESTICIDES AND OTHER AGRICULTURAL CHEMICALS IN CALIFORNIA, 1953-1963

		Systemic Poisonings			
Year	Total Conditions	Phosphate Ester Pesticides	DDT, Lindane, Endrin, Dieldrin	Other Agricultural Chemicals	Total
1953	377	146	9[a]	46	201
1954	391	101	1[a]	20	182
1955	531	126	4[a]	53	183
1956	789	197	11	73	281
1957	749	189	12	51	252
1958	910	227	14	87	328
1959	1,093	407	10	82	499
1960	975	283	7	78	368
1961	911	194	14	60	268
1962	827	140	21	58	219
1963	1,013	267	14	64	345
Total	8,566	2,277	117	672	3,066

(a) DDT only.

California employs about 20 percent of the pesticides used in the United States but accounts for about 7 percent of the nation's deaths due to these poisons. During the years 1951-1953, 128 people died in California from pesticides and other agricultural chemicals. Seventy-six of these were children and 29 were workers. Twenty-three others died. Arsenic accounted for 44 of the childrens' deaths and organophosphates, 13. All other pesticides accounted for 19. Twelve of the 26 workers who died were poisoned with organophosphates, 7 died from methyl bromide, and 10 from other poisons. Suicides now outnumber accidental deaths due to pesticides.(33) There were no deaths identified with DDT.

In southern Florida, deaths due to pesticide poisoning have been responsible for 9 to 10 percent of all deaths from poisoning and pesticides and have accounted for nearly 50 percent of deaths due to poisoning amoung children.(34) One hundred twenty-one deaths were attributed to pesticides in 1956-1967 among a population ranging from 950,000 in 1960 to 1,200,000 in 1967. Suicides included in these deaths accounted for 51 to 72 percent of the poison deaths. Deaths occurred in all age groups, but children under 5 accounted for 50 percent of accidental deaths due to poisoning, 86 percent were from organophosphates. Regarding agents involved, over half of all deaths were due to organophosphates and 10 percent involved the chlorinated hydrocarbons. Only three deaths were due to occupational accidents; all involved parathion. In the years 1964-1967, there were 61 cases of documented accidental pesticide poisoning, of which 11 were fatal. Seventy-two percent of the accidental deaths and the one occupational death were due to organophosphates.

The number of accidental ingestions of pesticides by children under 5 years of age reported by Poison Control Centers in 1968 was 3965 (Table 7). The total incidents reported involving pesticides were 5739 out of a total of 105,175 poison incidents reported.(35) This reference presents further breakdown by various categories (Tables 8, 9, and 10), but does not identify the halogenated hydrocarbons as a poison involved.

Although not recorded in the foregoing statistics, a large fraction of the occupational exposure and accidents occur in the agriculture industry. This industry has an unimpressive safety record in other aspects as well, and many preventable accidents and deaths have occurred since the halogenated hydrocarbons were first used in 1945. Most of the serious poisonings have resulted from disregard of safe practices. Aerial application has become the dominant method because of speed and efficiency. One might expect that accidents involving the aircraft itself and the pesticide load would be prevalent. In the 6 years ending in 1965, there were 322 occupational sickness cases in California involving employees of aircraft operators.(36) Eye conditions, chemical burns, systemic poisoning and "other conditions" are the categories of sickness. Of the 322 incidents, 220, or nearly 70 percent, involved systemic poisoning. Organophosphates caused 87 percent of the systemic poisoning cases. The author is careful to point out that these were the reported incidents and that many others occurred but were unreported. During the 6 years, aerial applicators treated 38 million acres. Several case histories reviewed emphasized that carelessness and lack of proper instruction and supervision were present in nearly every instance. Inadequate equipment was also a factor in these cases. In some instances, the worker carried contaminated clothing into homes where children were subsequently poisoned.

Flying accidents might be expected to be a significant cause of pesticide poisoning since crashes are frequently violent because of low altitude flown. In accidents in which the pilot was killed in the crash, contact with the pesticide would, of course, be

TABLE 7. ACCIDENTAL INGESTIONS AMONG CHILDREN UNDER 5 YEARS OF AGE REPORTED BY POISON CONTROL CENTERS, 1965-1968

Type of Substance by Year of Report

Type of Substance	1968		1967		1966		1965	
	No.	%	No.	%	No.	%	No.	%
Medicines	38354	53.6	38360	52.8	34670	53.6	34483	54.4
Internal	35516	49.6	34081	46.9	31213	48.3	30870	48.7
Aspirin	15523	21.7	16887	23.2	16076	24.9	16328	25.8
Other	16477	23.0	17194	23.7	15137	23.4	14542	22.9
External	4596	6.4	4279	5.9	3457	5.3	3613	5.7
Cleaning & polishing agents	10349	14.4	10414	14.3	9398	14.5	9343	14.7
Petroleum products	3573	5.0	3337	4.6	3243	5.0	3073	4.9
Cosmetics	4286	6.0	4639	6.4	3785	5.9	3271	5.2
Pesticides	3965	5.5	4087	5.6	3715	5.8	3856	6.1
Gases and vapors	64	0.1	102	0.1	96	0.2	87	0.1
Plants	2740	3.8	2890	4.0	2153	3.3	2028	3.2
Turpentine, paints, etc.	3947	5.5	3663	5.0	3260	5.0	3095	4.9
Miscellaneous	3688	5.1	4721	6.5	3911	6.1	3766	5.9
Not specified	597	1.0	448	0.6	403	0.6	350	0.6
Total	71563	100.0	72661	100.0	64634	100.0	63352	100.0

Source: Individual reports submitted to the National Clearinghouse for Poison Control Centers
1968: 71563 from 420 centers in 43 states(1); 1967: 72,661 from 395 centers in 43 states(1);
1966: 64,634 reports from 356 centers in 41 states(1); 1965: 63,352 reports from 341 centers in 40 states(1).

(1) Includes District of Columbia, Canal Zone, and Military Bases abroad.

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TABLE 8. CATEGORIES OF SUBSTANCES MOST FREQUENTLY INGESTED BY CHILDREN UNDER 5 YEARS OF AGE REPORTED BY POISON CONTROL CENTERS, 1965-1968

Type of Substance	1968		1967		1966		1965	
	No.	%	No.	%	No.	%	No.	%
Aspirin	15523	21.7	16887	23.2	16076	24.9	16328	25.8
Baby	10785	15.1	11336	15.6	10954	16.9	10854	17.1
Adult	1108	1.5	1141	1.6	963	1.5	1248	2.0
Unspecified	3630	5.1	4210	5.8	4159	6.4	4226	6.7
Soaps, detergents, cleaners	3501	4.5	3138	4.3	2582	4.0	2661	4.2
Vitamins	2748	3.8	2567	3.5	2093	3.2	1914	3.0
Miscellaneous internal medicine	2365	3.3	2641	3.6	2393	3.7	2474	3.9
Plants (excl. mushrooms and toadstools)	2339	3.3	2336	3.2	1707	2.6	1547	2.4
Bleach	2013	2.8	2164	3.0	2271	3.5	2276	3.6
Insecticides (excl. mothballs)	**1858**	**2.6**	**2120**	**2.9**	**1960**	**3.0**	**2056**	**3.2**
Disinfectants, deodorizers	1776	2.5	1805	2.5	1391	2.2	1162	1.8
Antihistamines & cold medicines	1758	2.5	930	1.3	864	1.3	838	1.3
Perfume, toilet water	1663	2.3	1720	2.4	1365	2.1	1167	1.9
Tranquilizers	1636	2.3	1670	2.3	1321	2.0	1275	2.0
Hormones	1426	2.0	1399	1.9	1371	2.1	1311	2.1
Analgesics other than Aspirin	1286	1.8	1673	2.3	1455	2.3	1193	1.9
Liquid polish and wax	1216	1.7	1422	2.0	1368	2.1	1364	2.2
Glues and adhesives	1208	1.7	1121	1.5	1039	1.6	900	1.4
Medicine combinations[a]	1177	1.6	--	--	--	--	--	--
Lighter fluids	1122	1.6	1078	1.5	1050	1.6	1022	1.6
Acids and alkalies	1034	1.4	965	1.3	959	1.5	930	1.5
Liniment	1021	1.4	896	1.2	758	1.2	703	1.1
Amphetamines	917	1.3	987	1.4	861	1.3	786	1.2
Rodenticides	914	1.3	916	1.3	801	1.2	814	1.3
Solvents and thinners	887	1.2	759	1.0	688	1.1	657	1.0
Miscellaneous external medicine	887	1.2	876	1.2	702	1.1	790	1.2
Laxatives	874	1.2	914	1.3	902	1.4	975	1.5
Antiseptics	861	1.2	856	1.2	742	1.1	756	1.2

(a) First year available, previously coded by the most toxic component.

Source: Individual case reports submitted to the National Clearinghouse for Poison Control Centers 1968: 71,563 reports from 420 centers in 43 states(1); 1967: 72,661 reports from 395 centers in 43 states(1); 1966: 64,634 reports from 356 centers in 41 states(1); 1965: 63,352 reports from 341 centers in 40 states(1).

(1) Includes District of Columbia, Canal Zone, and Military bases abroad.

TABLE 9. 1968 INGESTION REPORTS - BY CATEGORY

Product	Under 5 Years	Over 4 Years	Unknown Ages	Symptoms Under 5	Symptoms Over 4	Symptoms Age Unknown	Hospital Under 5	Hospital Over 4	Hospital Age Unknown	Fatal All Ages
V2 Paint	846	82	129	45	25	24	17	3	--	1
V3 Turpentine	752	97	54	105	19	5	9a	11	1	--
V4 Solvents and Thinners	887	201	148	137	60	33	105	26	2	4
V5 Oil paint, stain, and preservative	254	30	42	17	10	11	10	1	--	--
V6 Glues and Adhesives	1,208	151	110	69	37	10	16	7	--	--
XX Unknown	597	784	140	48	331	17	50	366	12	11
X1 Veterinary products	395	43	40	25	11	5	8	3	--	--
X4 Pesticide combinations	6	5	7	1	3	1	1	1	--	--
X5 Household product combinations	194	89	56	24	25	15	17	16	--	1
X6 Medicine combinations	1,177	3,461	222	157	1,849	79	129	1,774	30	32
X9 Miscellaneous products	906	238	177	64	48	31	11	10	--	1
02 Tobacco	217	1	29	8	--	--	1	--	--	1
03 Alcoholic beverages	55	82	11	21	48	6	10	30	2	1
04 Children's art materials	372	34	41	8	3	--	--	--	--	--
05 Venom - Snake or Spider	137	376	297	69	181	123	2	19	--	--
06 Ink	336	131	56	18	6	3	1	--	--	--
07 Matches	308	7	48	6	--	--	1	--	--	--
08 Fertilizers, Plant foods	312	66	53	12	5	7	3	--	--	--
09 Chemicals	650	420	243	51	76	36	46	37	2	7
10 Antiseptic medication	861	216	131	28	23	8	13	21	3	--
11 Liniments	1,021	223	100	81	75	15	44	52	4	2
12 Eye, Ear and Nose preparations	310	18	19	14	2	--	a	1	--	--
13 Ointments	423	22	40	11	2	1	1	--	--	--
14 Dental preparations	192	47	36	13	3	2	3	2	--	1
15 Camphor products	276	80	40	35	24	3	58	20	2	--
16 Medicated lotion and creams	152	29	21	4	5	2	4	3	--	--
17 Topical baby preparations	474	8	64	25	1	2	--	1	--	--
19 Miscellaneous external medicines	887	147	102	65	27	12	27	7	--	--
2X Antihistamines and cold medicines	1,758	337	81	144	120	12	59	63	2	--
20 Baby aspirin	10,785	449	216	339	36	6	542	17	5	3
21 Adult aspirin	1,108	778	89	61	182	15	79	158	5	8
22 Aspirin, unspecified type	3,630	1,404	101	143	286	7	233	387	5	1
23 Miscellaneous analgesics	1,286	1,655	186	95	533	40	54	452	11	10
24 Laxatives	874	113	47	79	11	2	23	--	--	--
25 Barbiturate sedatives	439	2,046	111	106	1,391	46	94	1,191	25	27
26 Nonbarbiturate sedatives	257	2,097	211	44	1,142	58	22	1,008	21	15
27 Cough medicines	773	101	48	96	28	4	25	6	--	--
28 Internal antibiotics	510	152	44	17	35	7	8	28	--	1
29 Psychopharmacologic agents	1,636	3,069	289	296	1,508	89	208	1,327	20	11
30 Anticholinergics, antacids	473	134	25	48	44	5	29	34	1	--
31 Diuretics	254	33	13	11	5	2	10	5	1	--
32 Iron preparations	393	38	25	37	3	3	96	a	2	1
33 Vitamins, minerals	2,748	338	151	79	18	--	25	--	--	--
34 Hormones	1,426	146	81	29	12	3	30	11	--	--
35 Cardiovascular medications	486	118	28	57	24	8	58	46	2	1

TABLE 9. (Continued)

	Product	Under 5 Years	Over 4 Years	Unknown Ages	Symptoms Under 5	Symptoms Over 4	Symptoms Age Unknown	Hospital Under 5	Hospital Over 4	Hospital Age Unknown	Fatal All Ages
36	Amphetamine type preparations	917	253	82	207	112	18	109	57	4	--
37	Antidiarrheal agents	337	25	15	51	2	1	31	3	--	--
38	Vermifuges	126	27	12	12	7	1	4	3	--	--
39	Miscellaneous internal medicines	2,365	1,208	265	228	472	48	121	369	9	7
40	Household bleach	2,013	310	194	141	62	23	67	34	3	--
41	Household disinfectant and deodorant	1,776	170	203	134	39	19	71	21	3	1
42	Corrosive acids and alkalies	1,034	280	180	410	112	53	253	72	5	3
43	Solid polish or wax	77	6	12	4	2	--	6	--	--	--
44	Dry cleaning fluid, spot removers	156	72	51	16	25	8	13	21	--	--
45	Soaps, detergents, cleaners	3,101	325	436	269	70	65	73	22	1	4
46	Shoe preparations	267	18	22	16	3	2	3	--	--	--
48	Liquid polish or wax	1,216	81	118	148	12	8	160	8	4	--
49	Miscellaneous household products	515	56	82	25	6	3	8	3	--	--
50	Kerosene	635	34	29	220	8	6	247	8	3	3
53	Lighter fluids	1,122	64	81	185	17	9	135	11	3	--
54	Naphtha	6	2	2	2	--	2	2	--	--	--
57	Gasoline	745	294	94	154	72	14	131	21	2	1
58	Petroleum solvents and cleaners	273	46	33	36	14	4	27	6	--	1
59	Miscellaneous petroleum products	792	51	60	90	10	4	68	1	--	1
60	Fingernail preparations	626	35	73	30	7	3	4	2	--	--
61	Perfume, cologne, toilet water	1,663	49	100	71	4	5	6	--	1	1
62	Personal deodorants	215	19	38	17	2	4	1	--	--	--
63	Hair preparations, excluding shampoo	529	56	65	34	9	8	5	1	--	--
64	Cosmetic lotions and creams	627	48	58	30	8	2	4	4	--	--
65	Hair shampoo	234	17	23	10	2	1	--	--	--	1
69	Miscellaneous cosmetics	392	19	38	23	1	3	7	--	--	--
70	**Insecticides**	**1,858**	**611**	**450**	**170**	**186**	**95**	**160**	**133**	**7**	**11**
71	**Rodenticides**	**914**	**142**	**129**	**47**	**20**	**14**	**63**	**13**	**4**	**4**
72	**Fungicides**	**52**	**28**	**19**	**4**	**3**	**6**	--	**1**	--	--
73	**Herbicides**	**116**	**64**	**74**	**7**	**26**	**25**	**5**	**5**	--	**9**
74	**Moth balls**	**827**	**31**	**64**	**24**	**3**	**3**	**13**	--	--	--
75	Animal repellents	65	65	49	22	37	20	2	1	2	--
76	Insect repellents	127	19	17	19	7	3	2	1	--	--
80	Carbon monoxide	25	100	32	11	51	15	4	36	--	10
81	Gases, vapors, fumes	39	220	214	8	129	93	3	32	1	1
90	Mushrooms, toadstools	401	60	59	9	6	3	7	15	1	--
91	Plants	2,339	476	463	106	100	41	33	26	1	3
	Total all cases reported	71,563	25,477	8,138	5,832	9,923	1,390	4,129	8,083	212	202

Source: Individual poison reports (phone inquiries and treated cases) submitted to the National Clearinghouse for Poison Control Centers by 420 centers in 43 states.

TABLE 10. INGESTION REPORTS BY AGE AND MAJOR CATEGORY

	Total All Ages	Under 01	01	02	03	04	05-09	10-14	15-24	25-44	45-64	>64	Unknown Ages
Medicines	60,021	753	6,636	16,466	10,962	3,537	2,028	1,049	7,480	6,046	1,860	309	2,895
* Internal	54,082	416	4,993	14,677	10,345	3,327	1,814	961	7,306	5,902	1,754	245	2,342
* Aspirin	18,560	89	1,648	6,441	5,684	1,661	608	251	1,315	380	63	14	406
* Other	35,522	327	3,345	8,236	4,661	1,666	1,206	710	5,991	5,522	1,691	231	1,936
* External	5,939	337	1,643	1,789	617	210	214	88	174	144	106	64	553
Cleaning and Polishing Agents	13,110	596	4,543	3,515	1,206	489	445	145	341	253	155	68	1,354
Petroleum Products	4,363	97	1,530	1,342	420	184	167	100	140	63	16	5	299
Cosmetics	4,924	177	1,520	2,019	456	114	95	32	58	22	18	18	395
Pesticides	5,739	282	1,577	1,210	620	276	300	91	207	228	101	38	809
Gases and Vapors	630	20	10	11	13	10	40	23	89	100	52	16	246
Plants	3,798	219	697	904	599	321	343	79	65	22	17	10	522
Turpentine, Paints, etc.	4,991	175	1,867	1,286	429	190	183	84	153	93	36	12	483
Miscellaneous	6,081	355	1,209	1,170	612	342	565	285	269	168	91	20	995
Unknown	1,521	30	156	227	110	74	53	45	267	289	114	16	140
Total	105,178	2,704	19,745	28,150	15,427	5,537	4,219	1,933	9,069	7,284	2,460	512	8,138

*These figures are not reflected in the total line.

Source: Individual poison reports (phone inquiries and treated cases) submitted to the National Clearinghouse for Poison Control Centers by 420 centers in 43 states.

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incidental, but an injured pilot who may have easily survived the crash could potentially be fatally poisoned. Thirteen hundred and twenty-eight aerial application accidents during 1963-1966 were studied.[37] The authors note that 1,040,000 hours are flown annually in application of farm chemicals and seeds. From 300 to 400 accidents occur each year with 30 to 50 fatalities. This rate is second only to pleasure flying. Encouragingly, the accident rate declined during 1951 to 1966 from 51.7 to 27.2 per 100,000 flying hours. The fatal accident rate declined from 7.9 to 2.7 in the same period. Fires accompanied fatal crashes in 70 percent of the cases in 1965, and 40 percent in 1966, showing a wide variation from year to year.

Twenty fatal crashes of aerial applications in 1967 were investigated in more detail to determine any link between toxic chemcials and pilot-error accidents. In four cases no toxicological examination could be made. Eleven of the remaining 16 pilots had at least one abnormal test result. Eight of the 16 showed evidence of organophosphate exposure and five of these were compatible with excessive exposure. In one case the pilot was not critically injured in the crash but was splashed with tetraethyl pyrophosphate (TEPP) and mevinphos (Phosdrin) and died within 20 minutes.

Conclusions

This short review has attempted to bring together enough of the pertinent information regarding manufacture, formulation, storage, and use of pesticides with emphasis on DDT to permit a determination to be made of the impact of direct interface of DDT with man. The conclusions that can be drawn from this evidence are:

- DDT is a chemical of relatively low toxicity, but should be respected and good industrial and personal hygiene measures should be followed in using it.
- In the manufacturing, formulation, transport, and storage of DDT, rather ordinary precautions have permitted many tons of the material in all forms to be processed and handled safely. Fires and cross contamination in storage and transportation are the principal concerns.
- Gross failure to take ordinary precautions in using DDT is about the only cause of serious insult to man. Unfortunately, innocent children seem to be the ones who suffer and die from poisons through failure of adults.

THE INDIRECT PATHWAY

As previously mentioned, there are two pathways of exposure to pesticides. The direct pathway was discussed in the previous section. The second pathway, indirect pathway, is discussed in the following sections. The discussion of the indirect pathway will describe the critical routes of exposure through the different portions of the environment.

Distribution From Primary Sources

The objective of this section was to determine the approximate distribution of DDT per year, according to primary sources of direct application. As a starting point, the following quotation is appropriate: "Precise application rates are not always available on a country- or basin-wide basis, nor is the exact fate of each of the various compounds known."(38) Despite the apparent difficulty in making estimates, an approximation can be arrived at using several assumptions. The following discussion will show how the final figures were derived.

Using an article by Bailey and Hannum(39), it was determined that approximately 20 percent of all pesticides used in the United States is applied to areas in California. Then, a USDA report to categorize land uses in California was used.(40) The results are tabulated in Table 11.

TABLE 11. CATEGORIZATION OF CALIFORNIA LAND USE(39)

	Acres x 10^{-3}
Crop land (total)	11,841
Used for crops	8,983
Used for soil improvement crops and idle	805*
Used only for pasture	2,053
Grassland pasture and range (total)	23,280
Open permanent pasture on farms	20,450
Woodland pasture	2,830*
Forest land (total)	39,406
Forest land in farms	3,403*
Not in pasture and not in farms	36,003
Special uses (total)	15,100
Urban areas	2,882
Rural highways, railroads, and airports	1,189*
Rural parks	6,663
Wildlife refuges	174*
National defense, flood control industrial areas	3,800*
State-owned institutions and miscellaneous other uses	102*
Farmsteads, farm roads and lanes	290*
Land with low agricultural use value	10,557*
Grand Total	100,184

The types of land which probably did not have appreciable amounts of DDT applied were determined. These categories are denoted by an asterisk in the table. Then, using principally the Bailey and Hannum article, average application rates to the various land types were assigned. Table 12 reports these.

TABLE 12. DDT APPLICATION RATES BY LAND USE[39]

Land Use	Application Rate
Used for crops	1.25 lb/acre
Used only for pasture	1.25 lb/acre
Forest land	0.75 lb/acre
Urban areas	0.4 lb/acre

Since all land-use categories were not covered by these rates, certain assumptions were made. First, applications for crop land will be reported for land actually used for crops. Second, crop land used only for pasture will be reported under a general category of "pasture" along with grass land, pasture, and range. Third, rural parks will be included as part of forest land.

The 1964 figure for DDT use in the U. S., 39,082,000 pounds, was rounded to 40,000,000 pounds.[41] Then a number for urban areas was determined in the following manner: Bailey and Hannum reported that 45,000,000 pounds of pesticide per year are used in urban areas. Further, they report that about half of the total was insecticide. Then, based on a 1962 total production of 700,000,000 pounds of pesticides, the total amount of insecticides would be 350,000,000 pounds. In 1962, about half of the insecticide production, 167,032,000 pounds, was DDT. Therefore, it was assumed that 25 percent of pesticide use was DDT. Since California uses about 20 percent of the total, the amount of DDT used in urban areas of California was calculated as follows:

$$\text{Urban use (Calif.)} = \frac{45 \times 10^6}{4} \times 0.2 \cong 2.3 \text{ million pounds.}$$

The next calculation was made for crop land in California. The Fish and Wildlife Service, according to Robeck, et al, reports that approximately one-fourth of U. S. crop land is treated, to some extent, with pesticides every year. Therefore, the following calculation was made for California crop land:

$$\text{Crop-land use (Calif.)} = \frac{8,983,000 \text{ acres}}{4} \times 1.25 \text{ lb/acre} \cong 2.8 \text{ million pounds.}$$

It is known that total DDT use in California is 20 percent of 40 million pounds or 8 million pounds, with 2.3 million pounds estimated for urban use and 2.8 million pounds for crop-land use. Hence, the remaining 2.9 million pounds must be distributed between pasture and forest use.

Therefore, of the remaining 2.9 million pounds, an approximate distribution will be 1.4 million pounds for pasture and 1.5 million pounds for forests. National figures can be arrived at from the knowledge that California use is about 20 percent of the total U. S. use. Table 13 summarizes the data presented. If the above assumptions are reasonably sound, then approximately 52.5 percent of the DDT used in the United States per year is used in agriculture, 18.8 percent in forestry and 28.7 percent in urban areas. Table 14 shows the DDT production and use within the United States from 1962 to 1967.[41]

TABLE 13. DISTRIBUTION IN 1964 OF DDT (MILLION OF POUNDS)

Land Use	California	United States	Percent
Crop land	2.8	14.0	35.0
Pasture	1.4	7.0	17.5
Forest	1.5	7.5	18.8
Urban	2.3	11.5	28.7
Total	8.0	40.0	100.0

TABLE 14. POPULATION EFFECTS ON SYNTHETIC ORGANIC PESTICIDES PRODUCTION[41]

Year	Production, 1000 lb	Lb Produced/ Person in United States
1962	729,718	3.909
1963	763,477	4.031
1964	782,749	4.074
1965	877,197	4.508
1966	1,013,110	5.145
1967	1,049,663	5.272

DDT in the Atmosphere

The primary source of pesticide contamination of the environment is in the process of application. This process of application is accomplished by spraying or dusting, and a portion of the pesticides applied can remain in the atmosphere and not reach the intended target. Thus, a portion of the pesticide can drift in the air mass and come in contact with non-target forms of life.

The significance of the atmosphere as a reservoir or compartment for DDT must be inferred from extremely tenuous data regarding the nature and amount of the primary source — the transmigration of small particles from soil and existing air concentrations. Very meager information is available regarding the nature and behavior of the droplets and particles in spraying or dusting of crops or their subsequent fate in the atmosphere.

We have attempted to estimate the entrance of DDT into the air from within the continental United States and to estimate its persistence or rate of removal. The DDT in the atmosphere is that which has a relatively low deposition velocity and is assumed to consist of very small primary particles of the compound, or more likely, natural aerosols in the atmosphere to which DDT has become attached. Very little is known about the air concentrations and even less concerning the discrete nature of the airborne material.

The principal sources giving rise to the DDT contamination of the atmosphere are

(1) Forms of the compound released during various applications to forests and crops

(2) The resuspension of fine dust particles containing DDT earlier deposited

(3) The possible evolution of DDT from codistillation from surface waters.

Spraying and dusting of crop land and water are carried out in such a way as to get as much of the pesticide on the "target" as possible. Some is unavoidably "lost" to the atmosphere in the immediate vicinity where diffusion processes dilute the airborne material to a low level. A large fraction of the pesticide applied reaches the target on the ground or other ground cover in the vicinity of the crops.

Studies[42] have compared the drift of spray and dust formulations. Under the same climatic conditions at an application rate of 1 lb per acre, the spray resulted in a concentration of 0.1 ppm DDT 2,640 feet downwind and the dust 1.5 ppm DDT at the same distance. It was concluded that this effect was due mainly to the difference in particle size between the two formulations. The dust particles have much smaller size than spray droplets.

The effect of particle size on drift has also been studied.[43] It was found that few particles less than 5 microns in diameter fall on the target area, and they can drift for many miles. Particles 10-50 microns in diameter were found deposited several miles from the source. Particles 100 microns in diameter exhibited little drift hazard under normal conditions. Particles greater than 200 microns in diameter were found deposited a short distance downwind. A similar study[44] showed that when dust particles were released 10 feet above the ground into a 3-mph wind, 2-micron-diameter particles traveled 21 miles, 10-micron particles 1 mile, and 50-micron particles 200 feet.

The studies mentioned above indicated that the greatest potential hazard to the general population comes from the particles less than 5 microns in diameter. Also, dusting contaminates the air much more than spraying. Spraying, however, is much more inefficient than dusting because of the larger particle sizes in the spray.

Thus, although wind-drift deposition during application can be significant to crops adjacent to the field sprayed, from a health point of view, deposition of the wind-drifted materials in the vicinity is not of concern, but, rather, that which does not deposit but continues to rise in the atmosphere and may travel great distances is cause for greatest concern.

Determination of DDT residues on 18 samples of ambient air collected over four California cities showed all but two contained measurable amounts of DDT.[45] Air samples from nine different stations in the nationwide air sampling network contained from 2×10^{-14} g/m^3 to 3.4×10^{-10} g/m^3.[46,47] Tabor's results for DDT measurement in air in agricultural communities showed DDT present in ambient air from less than detectable up to about 2×10^{-8} g/m^3.[21] Each of the communities where samples were taken were essentially in the center of extensive acreages of peaches, cotton, fruit

or vegetables. One of the communities included a formulating plant. Samples were taken generally during the pesticide application period.

The foregoing data appear to be the extent of air samples taken for DDT, leaving a wide range of concentrations from which to select a value for determining the atmospheric burden of DDT. A low value, 1×10^{-13} g/m^3, for determining the atmospheric burden of DDT has been used and it is anticipated that the actual concentration in local regions may exceed this by as much as a factor of 10^5, but the average may be more nearly 1×10^{-10} g/m^3.

The volume of air over the continental United States, to a height of 3000 meters, would contain approximately 3.60×10^5 g of DDT when the concentration is 1×10^{-10} g/m^3. The return of this airborne DDT to earth must be at the same rate the material is introduced into the atmosphere considered. The average deposition rate per unit area can be estimated by dividing the rate by the area. The DDT present in the atmosphere at any time is given by

$$M = \frac{K}{\lambda}(1 - e^{-\lambda t}) \quad ,$$

in which

M = mass present at any time = (V) (C),
t = time from start of introduction into atmosphere,
K = rate of injection,
λ = rate of removal = $0.693/T_{1/2}$,
$\lambda = 0.693/0.0823 = 8.4\ yr^{-1}$. [$T_{1/2}$ is taken to be 30 days (0.082 yr). This is approximately half-time for removal of fallout from troposphere.]

At equilibrium

$$M = \frac{K}{\lambda} \quad .$$

For a concentration of 1×10^{-10} g/m^3,

$$M = 3.6 \times 10^5 \text{ g.}$$
$$K = M\lambda,$$
$$K = 3.6 \times 10^5\ (8.4),$$
$$K = 3.02 \times 10^6 \text{ g/year.}$$

The deposition to the earth's surface for a concentration of 1×10^{-10} g/m^3 would be 3.86×10^{-7} g/m^2/year.

The estimated atmospheric input under the air concentration assumption as a percentage of the total annual U. S. use of DDT in 1964 (4×10^7 lb) is 0.017 percent.

Many assumptions have been made with no attempt to justify them, since data are not available to give better than educated guesses. The transport and deposition from applications beyond the perimeter of the United States have been deliberately avoided,

but could make some difference, although the difference would likely not be significant at the level of confidence of the several assumptions made.

The use of the 30-day half-life for removal of DDT in the atmosphere can be rightfully questioned. If anything, it is felt that the removal half-time could be shorter. The half-life of 30 days is that for small particles injected near the top of the troposphere; the actual removal time from lower regions could be considerably less than the 30 days used.

DDT is converted to other compounds in the presence of ultraviolet light[48], hence, any degradation from this cause would reduce the equilibrium concentration and call for a greater input to maintain the same concentration. The data for degradation in ultraviolet light are not applicable in any quantitative sense to the sunlight. This is particularly true since we have no information on the form of the DDT in the atmosphere, and since the ultraviolet light degradation is dependent on the solvent in which the DDT is dissolved. Only meager rate data were obtained in the work reported in the reference.

Codistillation of DDT from water surfaces could be a source of the persistent airborne concentration of DDT. One study showed that 30 percent of DDT codistilled in 20 hours at 26.5 C.[49] Release from water surfaces would enhance the airborne concentration, but the ultimate significance of this input to the global atmosphere is only speculation, since so very little is known about the influence of the form of DDT, water quality, and temperature on the rate. Relative water surface to land surface within the United States would indicate that, because the land surface is so predominant, the release from water surfaces could be disregarded. However, when the surfaces wet with precipitation at any time are considered, the ratio of wet to dry could be greatly altered.

In a similar manner, the resuspension of very fine dust containing DDT will continue to place this material in the atmosphere. A rough estimate can be made of airborne DDT from this source. If we assume that the total DDT used in the United States has been deposited in the northern hemisphere between latitude of 30°N and 60°N and that half yet remains, then some 1×10^{11} g or 1×10^{-3} g/sq meter on the average is available for resuspension.

Airborne dust can vary in concentration over a wide range depending on location and atmospheric turbulence. Local measurements show that, over desert terrain in the first few meters above the ground, average dust loads can be of the order of 1 mg/m^3.[50] Other data from nonurban areas show total dust loads more nearly 0.5 to 0.1 mg/m^3.[51] If we assume 0.1 mg/m^3 is airborne from the first 3 inches of soil, and further assume that the 1×10^{-3} g DDT/m^2 is proportionately airborne, then the airborne concentration of DDT from resuspension should be $\sim 1 \times 10^{-12}$ g/m^3. This concentration is in the range of that measured in the few air measurements available. This calculation suggests that resuspension may account for a sizeable fraction of DDT in the region near the surface.

The foregoing discussions are largely speculation, since facts are so few regarding the behavior of DDT. Most important uncertainties are the following:

(1) Whether the few air concentrations measured are typical of the United States.

(2) The vapor-particle nature of the material made airborne is not known.

(3) Variation of concentrations with elevation in the atmosphere.

(4) The half-life in air due to radiation and oxidation is not known.

DDT in the Soil

Pesticide applications have led to a buildup of certain pesticides in the soil upon which the crop has been raised. In particular, DDT soil residues may be important because of the number of crop lands treated, the level of DDT accumulated in the soil, and the potential redistribution of DDT from soil into other pathways. DDT can reach man by the uptake of DDT into the crop plant, leaking into water supplies, volatilization into the air, and by entrainment into the air on dust particles. Ingestion of DDT-containing dirt on foods and other objects is also likely to lead to contamination.

The object of this section is to illustrate a potential critical pathway through the soil system for DDT. Much of the data is lacking and some general calculations based upon general patterns of pesticide and land use have been used.

The year 1964 has been taken as the base. DDT manufactured in the United States during that year was estimated to be 18.14×10^6 kg[41]. About 9.53×10^6 kg of DDT was actually used in agriculture. Forestry still used large quantities of DDT and a value of 3.4×10^6 kg for U. S. forestry was assumed. Urban use patterns are difficult to determine; agriculture and forestry were assumed to represent the major contributors to DDT in the environment.

It is a reasonable further assumption that all of the DDT applied to agricultural or forested lands eventually enters the soil system.[52] It is further assumed that the DDT entering the soil system is the sum of the agricultural and forestry use or about 12.93×10^6 kg for the base year of 1964.

Surface runoff forms an important part of the pathways of redistribution mechanism. Wide variations exist in the reported values for the DDT content of surface runoff. A value of 3.0 μg/l[53, 54] appears to be a likely value and will be taken as the average. It should be immediately recognized that the measurement of pesticide content of agricultural surface waters is at best a very spotty datum. The actual content of surface runoffs will vary depending upon local environmental conditions, soil, crop, and agricultural practices. An equally uncertain number is the U. S. average annual precipitation, which is taken here as 0.76 m.[55]

It is necessary to estimate the amount of land area applied. About 25 percent of the total U. S. crop land, pasture land, and forest land received pesticide applications or about 1.68×10 acre.[56] DDT amounted to 25 percent of the total pesticide used in 1964.[56] An average runoff coefficient for the United States of 29 percent is assumed and that DDT content of rainwater does not significantly add to the total DDT content of runoff waters is assumed.

With the above assumptions, the annual rainfall on DDT-treated land was calculated as

$$\frac{7.6 \times 10^{-1}\ M \times 4.0 \times 10^{3}\ M^2 \times 1.68 \times 10^{9}\ \text{acre} \times 6.26 \times 10^{-2} \times 1.02 \times 10^{3}\ l}{1\ \text{acre} \times 1\ M^3} = 3.27 \times 10^{14}\ l.$$

The runoff from DDT-treated lands was about

$$3.27 \times 10^{14}\ l \times 2.9 \times 10^{-1} = 9.54 \times 10^{13}\ l \quad .$$

Total DDT runoff was

$$\frac{3.0\ \mu g \times 9.54 \times 10^{13}\ l}{1\ l \times 1 \times 10^{9}\ \mu g/kg} = 2.86 \times 10^{5}\ kg \quad .$$

DDT is known to readily codistill.[57, 58, 59, 60] The annual codistillation of DDT is probably $2.0 \times 10^{-2}\ \mu g/M^2$. Then, the codistillation from treated lands of DDT is likely to be

$$\frac{1.68 \times 10^{9}\ \text{acre} \times 6.25 \times 10^{-2} \times 4.0 \times 10^{3} m^2 \times 2.0 \times 10^{-2}\ \mu g}{\text{acre} \times m^2 \times 1 \times 10^{9}\ \mu g/kg} = 8.40\ kg \quad .$$

There is evidence that DDT and other chlorinated hydrocarbon pesticides are not significantly mobile in soil.[61] Therefore, soil mobility will be disregarded.

Degradation within the soil system is known to occur. Under optimum conditions of soil pH, aeration, and texture, the half-life of DDT has been estimated as 3.0 years.[62] At normal application rates and over a period of several years, DDT degradation kinetics in soil are zero order.[62] Thus, the annual DDT degradation (assuming a rate of 16.5 percent/year) will equal

$$12.93 \times 10^{6}\ kg \times 1.65 \times 10^{-1} = 2.14 \times 10^{6}\ kg \quad .$$

In agriculture, the concentrations of DDT-treated above-ground and root crops are likely to be 2.0×10^{-2} and 1.0×10^{-1} ppm, respectively.[63, 64, 65] The total 1964 U. S. yield of principal above-ground and root crops was 1.89×10^{12} and 1.60×10^{11} kg, respectively, calculated from U. S. crop-yield data[66]. Then, assuming the same distribution of crop land and of DDT usage, we can calculate that

DDT removed in harvest of above-ground crops =

$$\frac{1.89 \times 10^{12}\ kg \times 6.25 \times 10^{-2} \times 2 \times 10^{-2}\ mg}{1 \times 10^{6}\ mg} = 2.36 \times 10^{3}\ kg$$

and that

DDT removed in harvest of root crops =

$$\frac{1.60 \times 10^{11} \text{ kg} \times 6.25 \times 10^{-2} \times 1 \times 10^{-1} \text{ mg}}{1 \times 10^{6} \text{ mg}} = 1 \times 10^{3} \text{ kg}$$

Figure 3, although representing gross approximations of the fate of agricultural and forest land-applied DDT, is useful in estimating the fate of this compound in the environment. It is evident that two mechanisms account for the bulk of DDT removal from soil. Surface runoff and soil degradation of DDT account for annual removal of approximately 2 percent and 17 percent of soil-applied DDT, respectively. Other mechanisms of removal are insignificant by comparison. Only soil degradation results in complete removal of the intact molecule from the environment. Thus, if the use of DDT in agriculture and forestry were terminated, removal of the molecule from soil would largely be a function of the soil degradation rate.

Current information regarding the degradation of DDT in soil has been summarized by Edwards[67] and is presented in Figure 4. Extrapolation of this curve indicates that negligible quantities of DDT would remain in soil after approximately 15 years if no additional applications were made and original application rates were not sufficiently high to retard the proliferation of soil microflora. The data of Eno[68] indicate that normal degradation will take place below an application rate as high as 250 lb of DDT/acre.

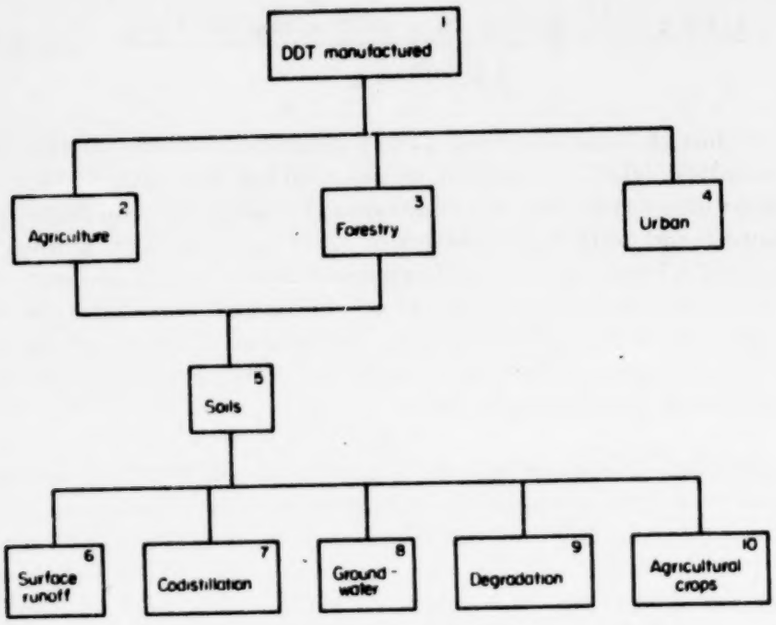
Restriction of DDT to essential crops would not affect, for practical purposes, the soil degradation rate, but it would have the effect of proportionally reducing the time required to achieve minimum acceptable levels of DDT in the environment.

Of further significance is the fact that DDT is strongly sorbed to the soil[61], and thus it is unlikely that groundwater supplies would be contaminated by movement of DDT through the soil profile. Due to its high affinity for soil particulate matter, it is probable that DDT is mobile in air or water largely as a suspended soil particulate-DDT complex.

Based on the above information, a suitable remedial program to reduce the quantity of agriculturally applied DDT in the environment might include three major objectives: (1) efficient disposition of threshold quantities of DDT on land and incorporation of residual applied DDT into the soil system, (2) retention of DDT in soil, and (3) attainment of the optimum practical DDT degradation rate in soil. In this manner, DDT might, with relative safety, be retained in the soil system until degraded to acceptable levels.

Blanket applications of DDT should be eliminated as an agricultural practice. Suitable application rates for given pests, crops, and soils should be devised and rigidly adhered to. Crop residues should be incorporated into the soil system where DDT decomposition may proceed rapidly under appropriate soil management.

Technology is presently available, but seldom fully utilized, for minimizing particulate removal from soil through suspension in the air or in irrigation and surface runoff waters. Improved tillage practices and crop management can increase water infiltration into soil. Grass waterways, settling basins, and flocculating and binding agents may be used to good advantage in reducing particulate transfer from soil to the atmosphere or to surface waters.



Legend for Figure

Code	Component of Process Description	Estimated Absolute Distribution of DDT for the U. S. (a, b)
1	Total manufactured	18.14×10^6 kg
2	Agriculture	9.53×10^6 kg
3	Forestry	3.40×10^6 kg
4	Urban	5.22×10^6 kg
5	Soil	12.93×10^6 kg (10.34×10^6 kg sorbed)
6	Surface runoff	2.86×10^5 kg
7	Codistillation	8.48×10^0 kg
8	Groundwater	Not detectable
9	Degradation in soil	2.14×10^6 kg
10	Agricultural crops	
	Above ground	2.36×10^3 kg
	Below ground	9.97×10^2 kg

(a) Based on 1964 statistics.
(b) Distribution of DDT in Components 4 to 10 based on a soil application rate (Component 5) equal to the sum of Components 2 and 3.

FIGURE 3. FATE OF DDT APPLIED TO U. S. AGRICULTURAL AND FOREST LANDS DURING 1964

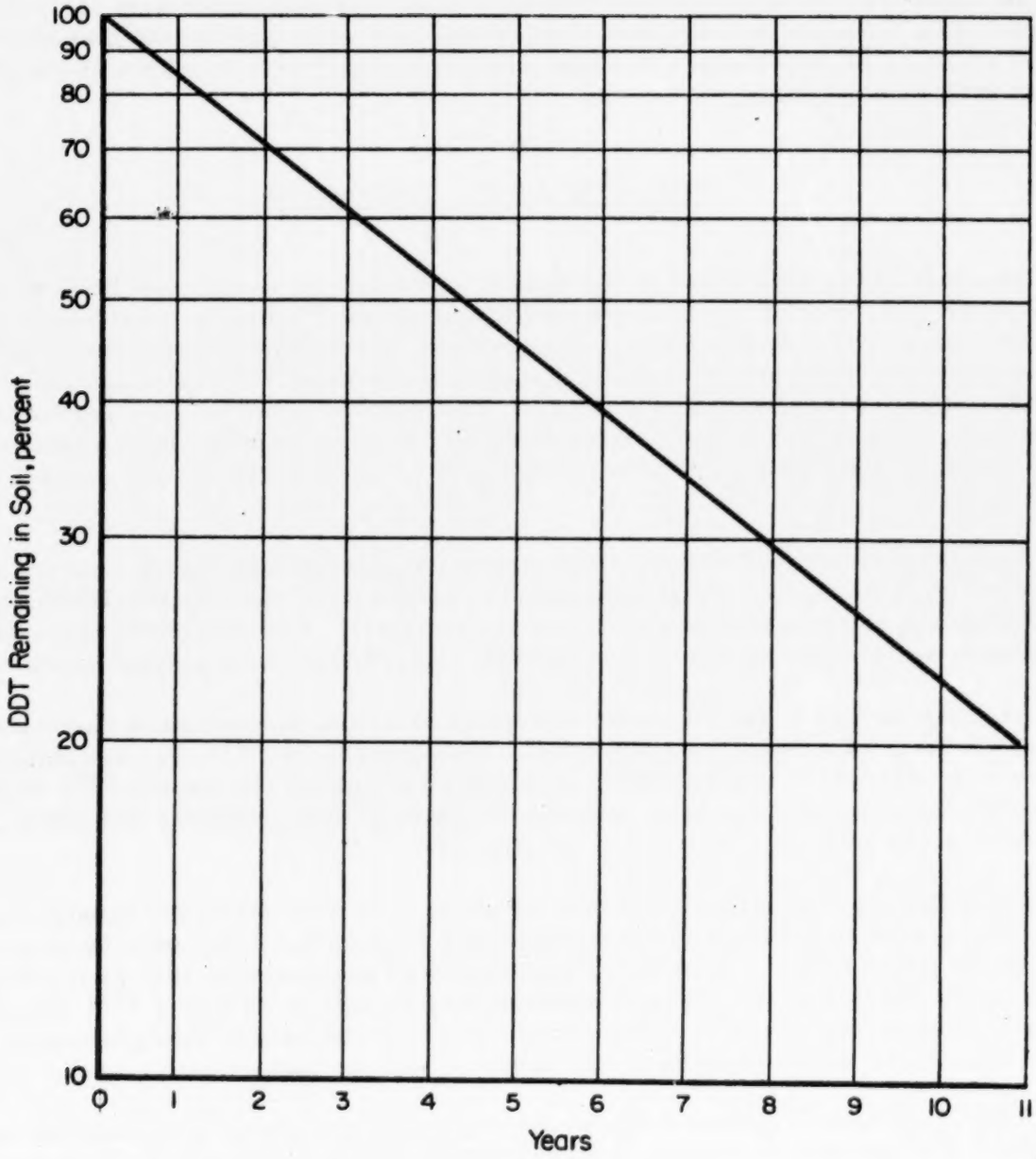


FIGURE 4. FATE OF DDT IN SOIL

Rapid soil decomposition of intact DDT occurs under anaerobic conditions[60]; however, with few exceptions practical agricultural use of the soil dictates that the soil be maintained in a generally aerobic state. Maintenance of proper levels of soil fertility, organic matter content, C:N ratio, and a favorable soil structure would maximize DDT decomposition rate under aerobic conditions.

In summary, the soil may function as a relatively safe repository for residual DDT resulting from proper levels of application, and with appropriate management, DDT in soil may be decomposed at rates which are significant in terms of the reduction of total DDT levels in the environment.

DDT in the Aquatic Environment

The bulk of the DDT found in the aquatic environment is carried there by surface runoff water from treated agricultural and forest areas (Figure 5). Extensive surveys in recent years reveal that most of the watersheds in the continental United States are contaminated with DDT and other chlorinated hydrocarbons.[69] Although the intended pesticidal effect of these chemicals is brief, the pesticide may be very persistent chemically. As a result, chemicals such as DDT may accumulate in the environment and be swept by runoff water into rivers and lakes and eventually to the estuaries and ocean.

In addition to surface runoff, DDT may be introduced into surface waters by direct application of aerial spray, by accumulation in falling rain, and from groundwater. The contribution via groundwater is apparently insignificant. Chlorinated hydrocarbons are not transported in significant amounts through surface soils into groundwaters.[70]

As much as half of the DDT that is applied by aerial spraying and dusting never reaches the target but becomes suspended in the atmosphere.[71] To this atmospheric reservoir is added DDT that codistills with evaporating surface waters.[20] Rain falling through the atmosphere can accumulate DDT-laden dust particles and suspended crystals to a concentration of 1 to 1 x 10^2 ppt.[72]

Codistillation represents one route by which DDT may leave the aquatic environment. The pesticide leaves aqueous suspensions in significant amounts by volatilization with the liquid phase. Laboratory experiments have revealed that as much as 50 percent of the DDT from a 0.01-ppm solution may be lost in 24 hours.[73] Since codistillation can be appreciable, it should be noted that the rate of this phenomenon is greatly affected by small changes in temperature and the concentration of the DDT.

Several items in Figure 5 include numbers. These figures are meant to indicate the orders of magnitude or ranges of magnitude of DDT concentrations reported in environmental studies. The DDT levels included in Figure 5 are those reported for the whole water sample, that is, for grab samples extracted and analyzed for pesticide content without attempting to separate suspended matter from the water. This is an important point. The solubility of DDT is very low, with the upper limit of solubility approximately 1 ppb. This means that a major portion of the reported DDT levels is actually that involved in the suspended matter in the water sample. Studies indicate that DDT is strongly sorbed to the soil.[61] Suspended sediments then account for the major vehicle for transport of this pesticide from treated land areas into the aquatic system.

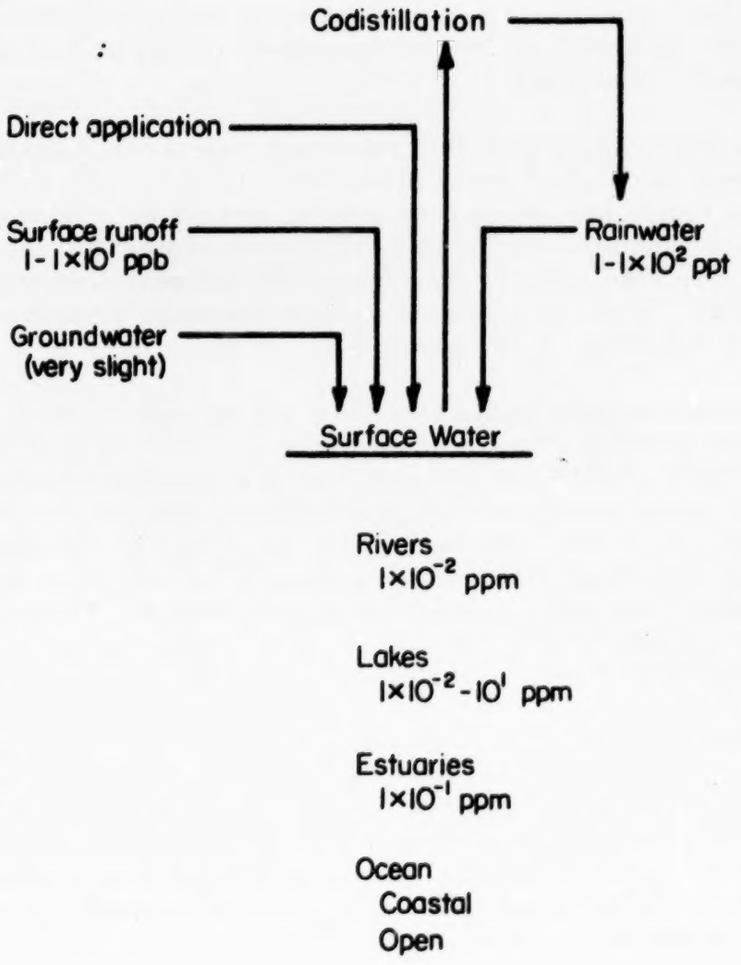


FIGURE 5. PATHWAY OF DDT TO SURFACE WATER

The category of surface waters has been broken down in Figure 6 into bottom sediments, suspended solids, and water. The DDT levels indicated here represent those reported for that compartment of surface water.

Chlorinated hydrocarbons such as DDT have very high partition coefficients; that is, these chemicals are 80 million times more soluble in oils than they are in water.[74] The consequences of this characteristic is two-fold. First, DDT can pass through a cell membrane with great facility, and, second, after having entered the cells of an organism, it will not be removed except by decomposition after death or by digestion following consumption by another animal. As a result of its tremendous solubility in lipids and chemical stability, DDT is accumulated in increasing amounts up through the levels of the food chain. Thus, the model of DDT in the aquatic ecosystem is essentially a reproduction of the aquatic food chain.

Once DDT has been transported into lakes and rivers, it readily enters the aquatic organism. The plants and invertebrates obtain DDT from all three compartments of surface waters: bottom sediments, suspended solids, and water. Animals, fish in particular, feeding on these contaminated plants and invertebrates concentrate the DDT content of many prey organisms. Fish may accumulate the pesticide material in concentrations of 1×10^2 ppm. Fish-eating birds become the most contaminated organism with body burdens of DDT reaching 1×10^3 ppm.

In addition to concentrating pesticides obtained through the food chain, fish may also obtain significant amounts directly from the water.[75] As was mentioned above, relatively small amounts of DDT are actually dissolved in water; however, a fish is in constant contact with water and passes large columns of water over its gills. Because of the extremely high partition coefficient of DDT for lipid and the ease of sorption onto hydrophobic surfaces, it is apparent that the gills offer an efficient mechanism for removal of the pesticides from water. Premdas and Anderson[76] found that, after 5 minutes' exposure to 1 ppm of DDT, Atlantic Salmon, Salmo salar, contained 1.56 ppm in the liver and spleen, and, after 1 hour of exposure, the level rose to 31 ppm.

It has been estimated that over 90 percent of the DDT body burden of the United States population is obtained from food.[63] The critical pathway to man through the aquatic environment is through man's aquatic diet, i.e., fish and other seafoods. Considering the average level of contamination of fish food, one can conclude that the average United States citizen obtains relatively little DDT from fish for the simple reason that the indivudual does not eat much fish. The per capita annual consumption of edible fish products in the United States is 11 pounds.[41]

In April of 1969, the Food and Drug Administration established the "action level" of 5.0 ppm for DDT residues permitted in fish shipped across state borders. Using this as the worst possible case and calculating from the per capita fish consumption, the United States citizen would ingest as much as 25 mg of DDT annually via fish foods. A very important point, however, is that very few people eat fish raw. Since the pesticide resides almost exclusively in the lipids, cooking should eliminate a considerable amount of the potential 25 mg. Studies have shown that frying, baking, or broiling fish eliminates a significant amount of the oils that contain most of the insecticides.[74] More realistic figures for levels of DDT observed in commercial fishes would be much lower than 5.0 ppm. Some reported levels are as follows: blue fin and yellow fin tuna, 0.1 to 0.6 ppm; anchovy, hake, mackerel, and English sole, 0.2 to 2.8 ppm.[77]

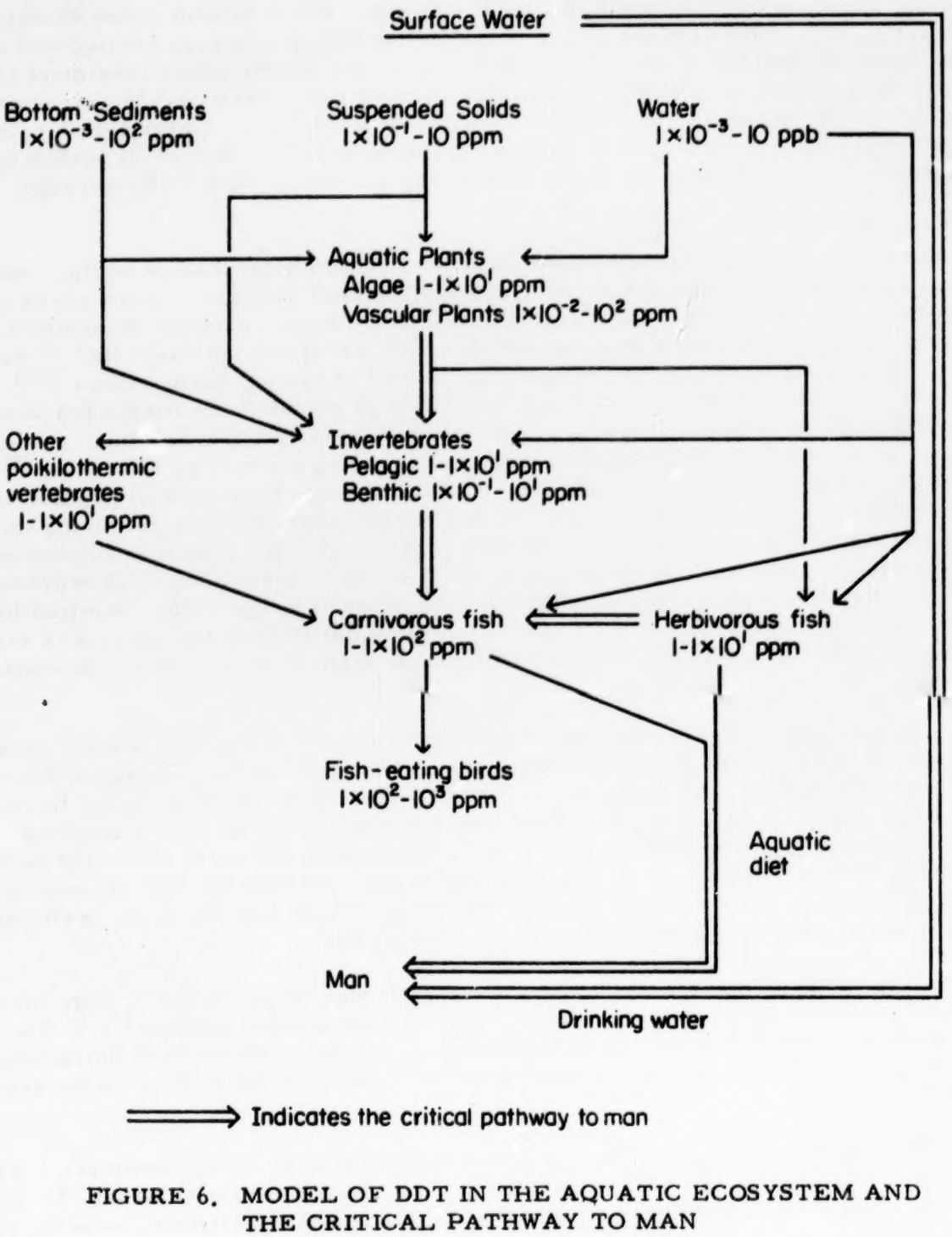


FIGURE 6. MODEL OF DDT IN THE AQUATIC ECOSYSTEM AND THE CRITICAL PATHWAY TO MAN

Water is really a part of man's food and therefore represents another potential dietary source of DDT. Surveys of the major United States river basins indicated an average of 0.023 ppb of the six major chlorinated hydrocarbons. Based on this concentration and the assumption that an individual drinks 2 liters of water every day, the average daily intake of DDT per individual is 0.046 ppb if the individual were drinking untreated water.[69] Most drinking water in the United States has been treated and research has revealed that 98 percent of the DDT is removed by the water treatment techniques most often employed. Either coagulation or filtration with a carbon slurry are capable of removing 98 percent of a 10-ppb contamination of DDT. Chlorination removes less than 10 percent of the insecticide from a similar concentration.[38] It seems unlikely, therefore, that drinking water represents any source of DDT to the average citizen.

As was noted, the main source of the aquatic environmental DDT is surface runoff water from treated areas. The pesticidal content of the soil in these treated areas can be very great. Once in the soil, the chemical is protected from photodecomposition and volatilization, except in the top few inches of soil.[78] Research indicates that 40 percent of the annually applied DDT still resides in the soil of two orchard studies.[79] The orchards concerned were treated with as much as 50 pounds of pesticide per acre per year for 20 years. A significant fact concerning chlorinated hydrocarbons revealed that they are immobile in soils, with only traces penetrating more than 12 inches.[80] Although it has not been conclusively proven, it is likely that the microbial populations of contaminated soils have been diminished or even eliminated, thereby removing the chance for metabolic removal of DDT and adding to its longevity. The significance of the chemical stability and soil relationships of DDT is that contaminated soil represents an extensive reservoir for the aquatic environment. Even if usage of the chemical immediately ceased, agricultural soils would continue to yield DDT to the watershed for 15 to 20 years. Each time the soil is tilled or eroded, a fresh source of contamination is released.

The majority of this report has been addressed to the direct effect of DDT on man via his aquatic diet and drinking water. An additional indirect manner exists in which DDT and other chlorinated hydrocarbons may affect man. This effect might be termed "environmental degradation", and its consequences are more subtle than something medical. Environmental degradation means the catastrophic loss of wildlife that have been poisoned, reduced reproduction capacity of fish and fowl that provide recreation for man, and unexpected resurgence of pests due to their insensitivity to the pesticide while its normally effective natural enemies are destroyed.

There are in the literature several accounts of sudden large losses of fish which can be correlated to sudden releases of pesticides into the aquatic system.[2,3] The impact of such occurrences on the sport fisherman is obvious. Heavy bird mortalities have also been reported and blamed on the high concentration of DDT found in the bird tissues.[4,81]

The "thin eggshell" syndrome has received a great deal of recent notoriety. Evidence indicates pesticides, and DDT in particular, may be the causative agent.[4] The shells of many birds' eggs have been proven fragile due to shell thickness, with the result a high breakage and loss of offspring to the bird population. A partial list of birds in which this has been observed would include the osprey (Pandion haliaetus), Bermuda petrel (Pterodroma cahow), American woodcocks (Philohela minor), peregrine falcon

(Falco peregrinus), golden eagle (Aquila chrysaetos), bald eagle (Haliaeetus leucocephalus), and herring gulls (Larus argentatus).

Many of these same species as well as additional species suffer a high mortality of chicks before and shortly after hatching. This is remarkably similar to effects reported for fish[1], and it may be that the mechanism of insecticide transfer from egg yolk may be similar in fishes as well as birds.

Observations of this loss of reproductive success in fishes and birds as well as several major fish kills have suggested that DDT is to blame. Although man may not be seriously threatened by his pollution of the aquatic ecosystem, he may stand to lose an aesthetic and recreational aspect of his life.

DDT in Foods

Disagreement exists as to the exact proportion of the total DDT intake from food. At first observation, it would seem that all of the pesticide intake should be via the food supply for all of those persons not directly involved in manufacture, transport, application of pesticides or in agriculture. Campbell[63] calculated that food contributes over 90 percent of the DDT absorbed by the general population. This calculation has been questioned recently because of the apparent differences in body burden between people living in northern versus southern states. The DDT body burden of the Negro population has been reported as nearly twice that of the Caucasian population.[82] Three possibilities exist:

(1) Southerners eat a substantially different diet than Northerners and that diet contains substantially higher levels of DDT and other pesticides.

(2) Food consumed in the South is no different but contains substantially higher levels.

(3) Dietary DDT is only a partial contributor to the DDT body burden.

The latter possibility seems to be more likely, since pesticide levels in foods are carefully controlled by both state and Federal monitoring systems, and it is unlikely that a continued higher residue level in southern states would go unnoticed. Further, food is a highly mobile commodity being transported over the entire United States. Dietary habits differ in the various regions of the United States, but the enormity of the U. S. food-processing industry is such that regional differences are unlikely to make substantial differences in dietary intake. It is more likely that the diet contributes only 50 percent and, as an upper limit, 90 percent of the body burden.

The lessening of the import of the diet as a prime source of DDT body burdens is suggested from other data, although there is no conclusive information. The worldwide level of DDT in peoples is remarkably similar.[86] Unlike the continental United States, there are large differences in dietary habits and regulation of pesticide residues. Food supplies between the continents are not as readily exchanged as within a given country because of the restrictions of trade, costs, distances, and a number of other factors.

The amount of DDT in food appears to be decreasing. For example, the amount of DDT in restaurant meals was found to be 0.038 mg in 1962-64 compared with 0.178 mg in 1954.[83] This decrease in dietary level of DDT occurred even though there was a continuing large-scale use of DDT during this period. The decrease in DDT consumption is not consistent with the constancy of the level of DDT in the body fat of the general population. Durham, Armstrong, and Quimby[12] and Hayes and Durham[14] have shown that the fat-storage level is a function of the dietary intake of DDT. Thus, if the storage and turnover rates suggested by these studies were correct, and if dietary DDT was responsible for 90 percent or more of the DDT intake, then one would have expected a decrease in the general population body burden. This contention is underscored in Table 15; DDT usage remained constant while DDT content of food decreased almost fivefold.

The DDT content of major food commodities is shown in Table 16. Meat is the largest single contributor to the dietary intake. Durham, Armstrong, and Quimby[12] demonstrated experimentally that products of animal origin (meats, gravies, meat combinations, and eggs) contained large quantities of DDT. Dairy products also contained large amounts. Hayes[3] showed that in 1958 the mean daily DDT intake of meatless meals was 0.041 mg. Walker[4] found the average meal in 1954 to contain 0.184 mg or about four times as much as the meatless meal. The critical pathway for DDT to man through the dietary patterns of the general population is illustrated in Figure 7.

Milk and dairy products are the next largest contributor to the dietary intake. DDT was found in the breast milk of 30 out of 32 women sampled, with concentrations of from 0.01 to 0.77 ppm and a mean of 0.13 ppm.[87] Market-basket surveys[83] indicate that fluid milk contains 0.04 ppm, as do manufactured dairy products.

The average levels of DDT in prepared baby foods from 684 random samples was less than 0.005 ppm from 1963-1967.[85] Thus, for the infant, it is reasonably certain that the major dietary source is milk and that breast-fed infants are subjected to higher levels than formula-fed infants.

In summary, DDT content of foods represents a major portion of the DDT intake of man. It is the major point of Government regulation. DDT levels in foods have decreased while the level of DDT usage remained constant. Having no evidence to the contrary, it is likely that the decrease in DDT content of foods was more a reflection of improved regulation than a significant alteration in agricultural or food-processing techniques. The largest single hazard to man from his diet appears to be meat and then milk. The dietary intake of infants could be significantly lessened if processed milk and formula feeding were universally instigated. There are serious conflicts in the evidence, and the assumption that all of the body burdens are due to food is open to question. The lack of response of the body burden to the decrease in the food supply indicates that other factors must play an important role in regulation of the body burden in man.

DEVELOPMENT OF THE POLLUTION CHAIN DIAGRAM FOR DDT

All of the foregoing discussion can be summarized in the pollution chain diagram shown in Figure 8. This diagram commences with the source in manufacturing and

TABLE 15. TREND OF DDT USE AND CONCENTRATION IN FOODS

Year	DDT Concentration in Foods Market Basket Studies, mg/kg body wt/day[a]	DDT Used in U. S., 1000 lb
1953	0.0025	52,952
1963	0.0005	76,958
1965	0.0004	50,369
1966	0.0005	50,435
1967	0.0004	21,583
1968	0.0003	--

(a) 70 kg man.

TABLE 16. DISTRIBUTION OF DDT IN THE MAJOR FOOD COMMODITIES AND INGESTION BY MAN IN 1967

Commodity	Mg Ingested Per Day	DDT, ppm	Mg DDT Ingested Per Day	Percent DDT Ingested
Meats	2.77×10^5	0.189	0.052	62.7
Manufactured dairy products	6.72×10^4	0.04	0.0027	3.25
Fluid milk	3.47×10^5	0.04	0.014	16.9
Fruits	1.96×10^5	0.025	0.0049	5.9
Vegetables	1.92×10^5	0.04	0.0077	9.2
Potatoes	1.45×10^5	0.002	0.0003	0.4
Grains	1.82×10^5	0.007	0.0013	1.5
Oils, fats, and shortening	4.5×10^4	0.006	0.0003	0.4
Sugar	1.21×10^5	Trace	--	--
Total			0.083 mg DDT/day	

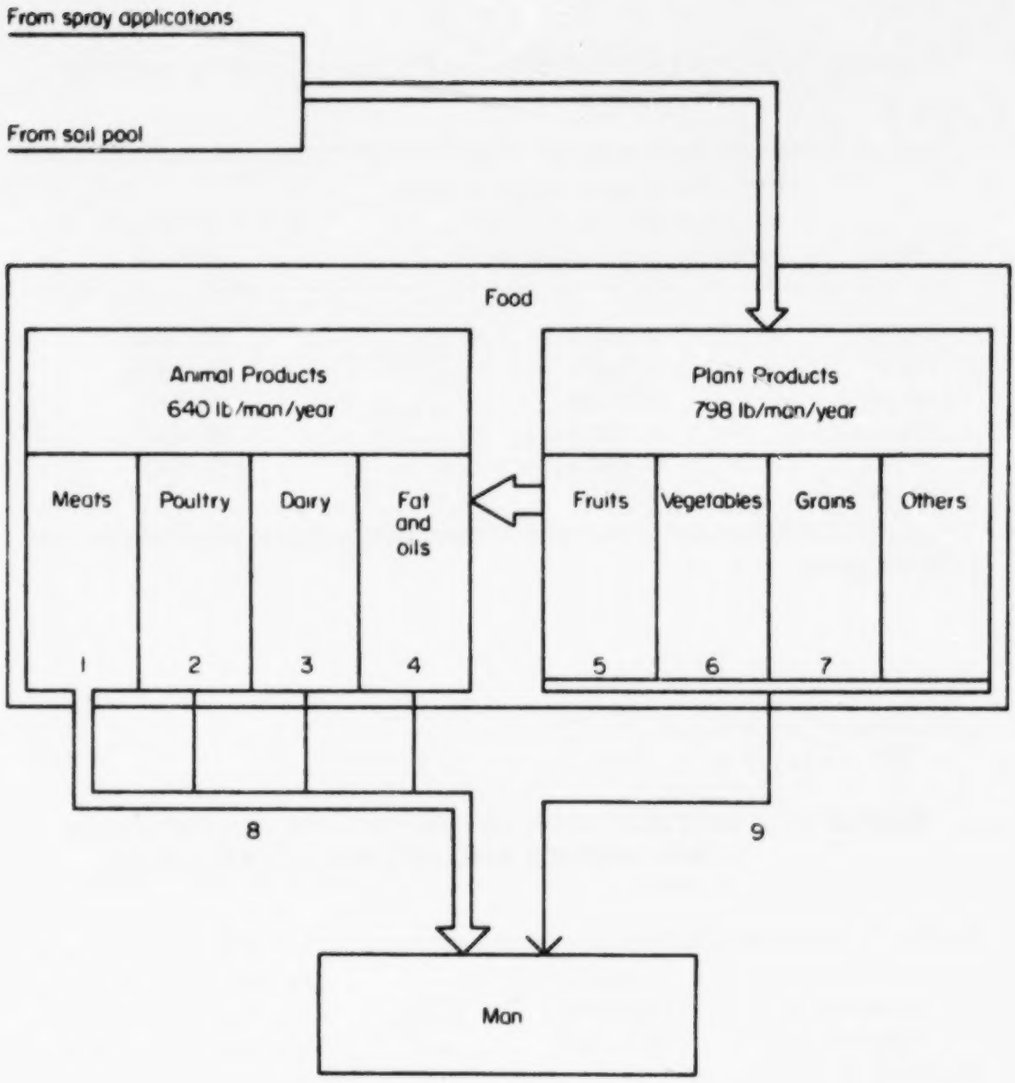


FIGURE 7. CRITICAL PATHWAY FOR DDT TO MAN THROUGH THE DIETARY PATTERNS OF THE GENERAL POPULATION

1. Total intake of meats and meat products was 157.7 lb/man/yr.
2. Total intake of poultry was 87.7 lb/man/yr.
3. Total intake of dairy products was 364 lb/man/yr.
4. Total intake of fats and oils was 51.3 lb/man/yr.
5. Total intake of fruits was 132.1 lb/man/yr.
6. Total intake of vegetables was 199.9 lb/man/yr.
7. Total intake of grains was 35.7 lb/man/yr.
8. DDT intake attributed to meat and meat products was 0.31 mg/man/day.
9. DDT intake attributed to plant products was 0.04 mg/man/day.

leads through the several pathways and compartments to man. The detailed critical pathways in each compartment are given in the respective foregoing sections. The legend to Figure 8 gives the rates and levels for the base year 1964.

Groundwater is not a significant compartment and is excluded from the critical pathways.

Land and Soil, Surface Water, and Food are the prime compartments leading to man for the general population. Surface Water does not contribute directly but indirectly through the food compartment to man's burden. Thus, the major pathways to man consist of direct applications to food and forage crops via soil residues. Of these, the meat and kindred products portion of the diet is the principal pathway. Thus, if effective new regulations or actions were needed to rapidly decrease the exposure of the general population to persistent pesticidal residues, particularly DDT, efforts should be directed here first. For a second segment of the population – infants – the critical pathway is more complex leading through human compartment thence to human milk. Residues in human milk are much greater than in cow's milk and in prepared baby foods. Should action be required to reduce the infant burden of these chemicals, then attention should be focused on the reduction of the intake of breast milk and the conversion of infants to formula and prepared foods.

In terms of the occupationally exposed, the important pathway in most cases is via inhalation. The air environment becomes the critical compartment, and attention to the concentration of these chemicals in the air becomes the critical feature for regulation and research in the working environment.

If the overall problem of the presence of these persistent compounds (modeled by DDT in the environment in general) is now considered, the pathways leading to a human health hazard are not critical. The detailed critical pathway for the aquatic environment is the dominant feature. The pathway through the aquatic environment is highly conservatory and the species within the system are relatively susceptible to chronic effects from DDT levels that are presently considered below the threshold level for human health. The actions necessary to protect the sensitive elements of the aquatic environment are thus not likely to be the same as those required to prevent the predictable human chronic diseases.

COLLECTION AND EVALUATION OF INFORMATION

The collection and evaluation of the information on pesticides has not proved to be an adequate test of EHS information systems, since the prime responsibility for pesticides has rested with FDA. During the course of this contract, FDA was removed from CPEHS and, hence, this important information system became an extra organizational component from an internal one. Our own independent literature search was relied upon to build up the data presented above. During the course of the exercise of the existing EHS information network, only a limited number of references and data sources were specifically identified with pesticides. These sources had already been covered by our independent literature search.

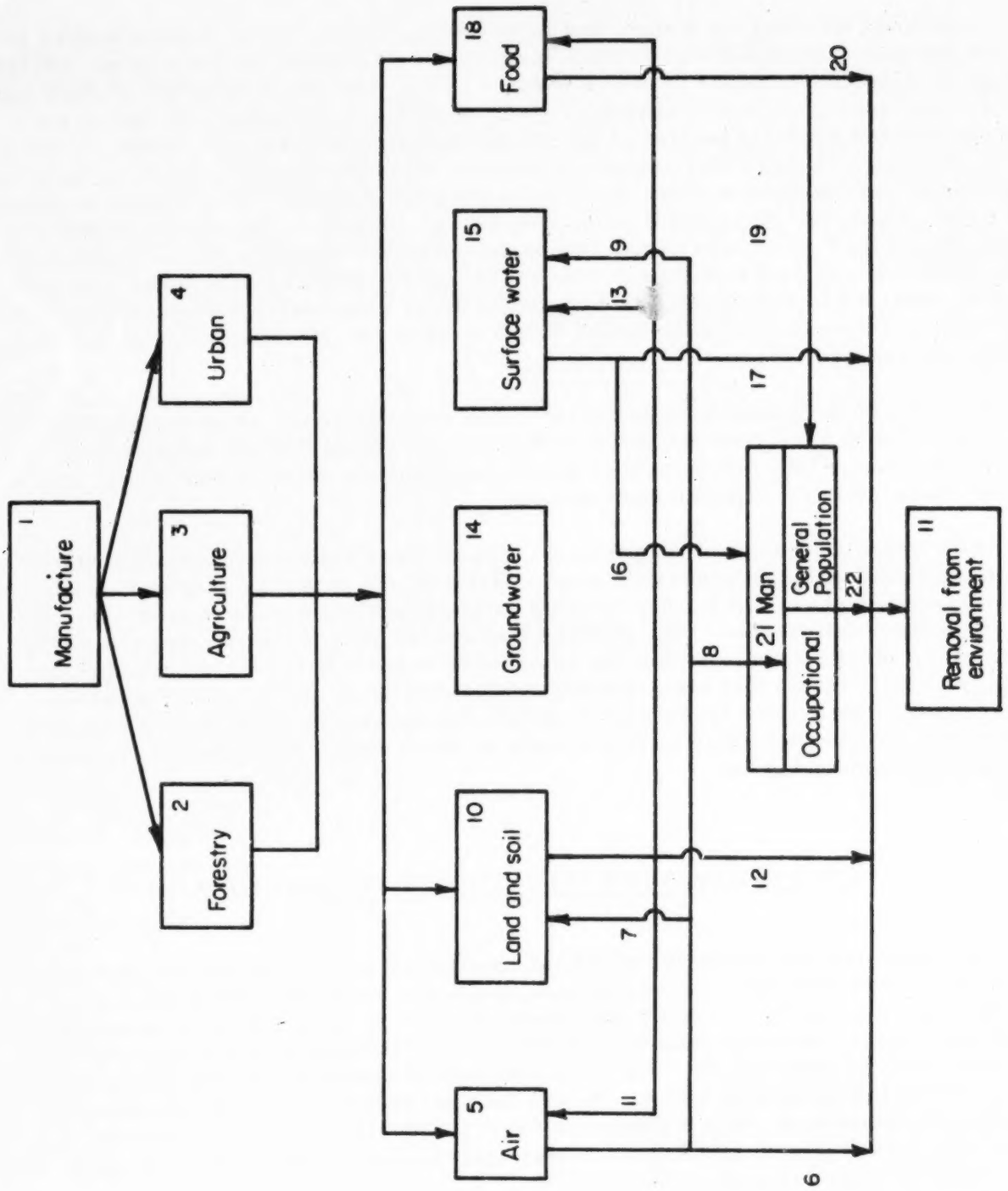


FIGURE 8. CRITICAL PATHS OF DDT IN THE ENVIRONMENT

Legend for Figure 8

Code	Component OP Process Description	DDT Concentration, degradation rate
1	DDT used in U. S. in 1964[38]	18.14×10^6 kg
2	DDT used in Forestry in 1964[39, 40]	3.40×10^6 kg
3	DDT used in Agriculture in 1964[39, 40]	9.53×10^6 kg
4	DDT used in Urban area in 1964[39, 40]	5.22×10^6 kg
5	Amount of DDT in atmosphere over U. S. at one time	3.6×10^5 g
6	Half life (T1/2) of DDT in atmosphere[23]	30 days
7	Deposition rate of DDT in atmosphere (1964)[23]	3.86×10^{-7} g/m^2/year
8	Inhalation of DDT from atmosphere[30] (a) In heavily sprayed areas (b) In large cities	 0.0002 to 0.0008 mg/man/day 0.000009 mg/man/day
9	Deposition rate of DDT in atmosphere (1964)	3.86×10^{-7} g/m^2/year
10	DDT applied to soil system in 1964[39, 40]	12.93×10^6 kg
11	Rate of injection of DDT in atmosphere over U. S. (1964)[23]	3.0×10^6 g/year
12	T1/2 of DDT in Soil System[62]	3.0 years
13	Annual DDT in runoff[61, 89, 90, 91]	2.86×10^5 kg
14	DDT in ground water[55]	Insignificant
15	Amount of DDT that reached estuaries and ocean from U. S. in 1964	2.63×10^4 kg/yr
16	DDT intake from drinking water[32]	0.000023 mg/man/day
17	Degradation of DDT aerbic water	T1/2 = 5.0×10^{-2}
18	Total DDT in foodstuffs	Data unavailable
19	DDT intake from food[83]	0.04 mg/man/day
20	DDT metabolized by general population	Data unavailable
21	DDT consumption general population	0.09 mg/man/day
22	DDT loss from body storage[14]	<0.3%/day

A list of data sources is appended to Volume II. Generally, the literature has been the most reliable source. Although a large number of references are available concerning pesticides, DDT in particular, there has not been an emphasis upon environmental effects. Largely, the data has dealt with acute rather than chronic effects. Further, the number of species examined has been highly restricted and conforms to the requirements for pesticide registration. In terms of human effects, the data seem to be adequate for the evaluation of the immediate health effects upon man. Since few or no true studies of chronic effects have been conducted with tools of sufficient sensitivity, it has not been possible to estimate the true effect of long-term exposure upon man. The total literature on environmental effects has been primarily about the persistent pesticides, and, for this very reason, they were chosen for our case study. Again, the information here becomes even more specialized to DDT alone.

The reason for a data deficiency can be understood by a historical evaluation of the literature. The earliest reports have dealt exclusively with the acute toxicity to man. Later reports dealt with the extensive series by Hayes and his coworkers of the ingestion of mg amounts by volunteers. Most of these studies still were designed with acute rather than chronic toxicity in mind. By this time, some concern appeared in the literature about the environmental aspects. There followed a long series of papers dealing with the acute toxicity to a number of nontarget wildlife, but again, not chronic studies. Observations then began to enter the literature that suggested there might be chronic effects. Simultaneously, there appeared studies showing that DDT and other chlorinated pesticides were ubiquitous. The latest papers deal with the pharmacological effects of these materials upon birds. Some of these, especially the apparent disruption of vitamin D and calcium metabolism, cause a great concern. No specific clinical finding can yet be found in the literature to suggest a similar disruption in man. Nor can we find any substantial evidence for carcinogenicity, yet rumors persist to this effect.

In sum, it appears that the proper experiments have not been designed and now the opportunity is lost, since no control population of man can be found upon which to base comparative epidemiological experiments. Much of the suggestive literature is found outside of the normal toxicology literature appearing in the general journals, Nature and Science. It would appear that the best monitor of potential problems in chronic human exposure is likely to be found in the ecological literature, since these papers preceded the actual experimental data showing a cause and effect relationship in the particular species. The identification and sorting out of the ecological effects and their classification prior to the advent of a concern for human effects is likely to be possible only in a judgmental manner by the use of two types of specialists: a specialist in the field of pesticides or other chemical stressor who keeps abreast of the literature specifically in that field and a specialist in general ecology who remains abreast of problems in the decline of specific species. These two specialists need to interchange observations and thoughts as to which ecological alterations are likely to be of significance to human health.

The bibliography specifically related to the development of pollution chain diagrams and models is cited in the above descriptive narrative.

IDENTIFICATION OF INFORMATION NEEDS

As with almost all other environmental stressors, the problem has been more readily recognized than the information base upon which possible solutions can be based. There are large areas in which only estimates of the problem could be made.

The most striking environmental problem is the lack of knowledge of the ultimate fate of most compounds in the environment. In the study, an arbitrary decision has been made that all non-DDT residues will be excluded from consideration. The pathway of DDT metabolism is complex, as is shown in Figure 9. Residue data have been reported only in terms of the toxicant or commercial chemical, DDT. Some scattered information is available on the primary metabolite, DDE. The later products of metabolism are generally ignored and hence not even analyzed. These later metabolites are not without biological activity, since they are known to induce specific enzymes in animals fed low levels.[88] Even though there is knowledge of the biological products, there is essentially no knowledge of the ultimate products formed by physical and chemical action in nature. What are the photodecomposition products? Distress is underscored by the realization that the most studied of all commercial chemicals, DDT, has been chosen. It has not been possible to delve into other classes of compounds sufficiently in this study, but it is likely that similar knowledge concerning other chemicals widely dispersed in the environment is also lacking.

Tremendous gaps exist in detailed knowledge of the fate of DDT and other pesticides in the aerial environment. Scattered references occur on the pesticidal content of air, yet little knowledge is available as to the controlling factors for pesticidal dispersion, the particle-size distribution, the inventory in the atmosphere and the mechanisms of reentrainment into other portions of the environment. Since there is so little knowledge, little confidence can be placed in the relative role of aerial contamination in the total health effects of these compounds. The aerial environment remains as one of the portions of the environment that is directly within the cognizance of EHS, and EHS study hence remains appropriate.

The impact of studies of the aerial transport is likely to be of indirect importance. As a health hazard, inhalation of pesticides away from the site of application is not a significant contributor to the human burden of pesticides. The aerial environment is most likely to affect wildlife and the total ecosystem prior to an effect on man. Further, the international aspects are clear, since aerial transport occurs over wider geographic boundaries and is more rapid than other modes of transport.

An area of potential deficiency outside the EHS realm is the fate of pesticides in the soil system. Some information is available suggesting that important ecological advantages could be obtained by the manipulation of soil chemistry (anaerobic degradation of DDT appears to proceed much more rapidly than aerobic). The sparsity of data suggests that a carefully prepared program is essential. The potential improvements in the general environment are likely to be great.

Specific exposure patterns within various groups in the United States are difficult to estimate. Robinson[86] has summarized the world human DDT and lindane residues and has found a remarkable uniformity. The uniformity may reflect an insufficient understanding of the variation or poor sampling procedures. Without more detailed information, it is difficult to predict those population groups likely to reach a hazardous position.

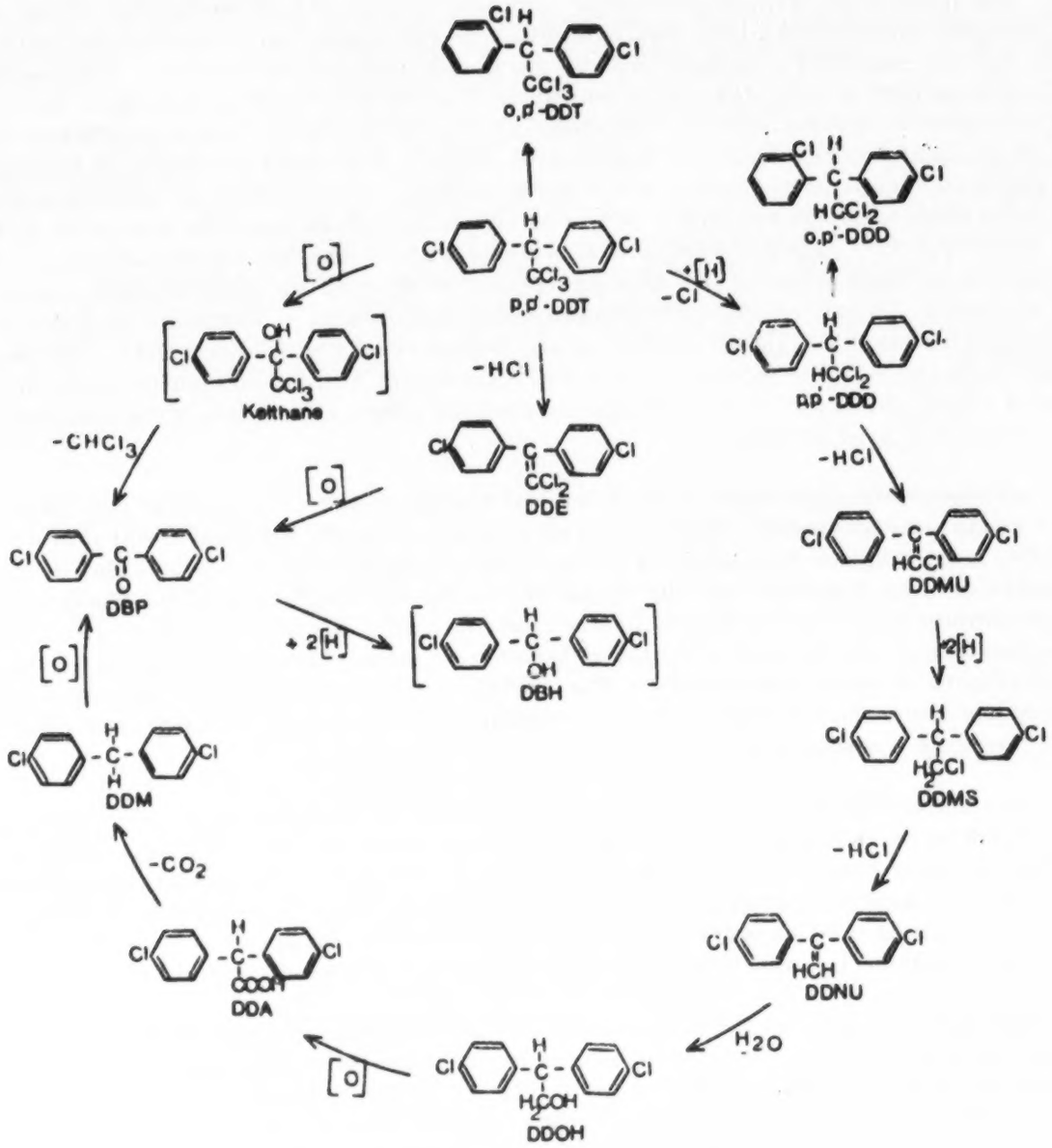


FIGURE 9. SUGGESTED DDT METABOLIC PATHWAYS

Overall studies on the metabolism of pesticides in man at chronic levels are also lacking. A question exists as to whether the rate of metabolism of these persistent compounds is dose dependent. The conversion of DDT to polar metabolites and their excretion from the body appears to decrease as the level of DDT decreases in the diet. In other words, below a certain level of DDT in the body, there does not appear to be any metabolism. While this statement appears self-evident, it could mean that a uniform and persistent background of DDT and its metabolites will remain until death. The cocarcinogenic or synergistic effects of this low-level residual portion of DDT are not known. Neither is there any information available in the literature to show that a given regime can increase, or for that matter decrease, the rate of metabolism of DDT. In terms of policy, the termination of DDT usage does not guarantee that the population will return to a "zero-DDT" state until the presently exposed generation is deceased. Latent effects of DDT or other persistent pesticides are likewise unknown.

In summary, while the oldest and most widely used pesticide, DDT, was studied, large areas were found where information is not available. The areas of information need are generally now outside the EHS realm. Aerial transport and decomposition of pesticides by physical and chemical factors in the environment are the notable exceptions of data need remaining in the EHS realm. Surveillance of the literature and information sources for likely indicators of future environmental threats from pesticides appears most feasible from literature outside of toxicology-of-pesticide research. A method for sorting ecological indicators relevant to human-health hazard is needed.

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